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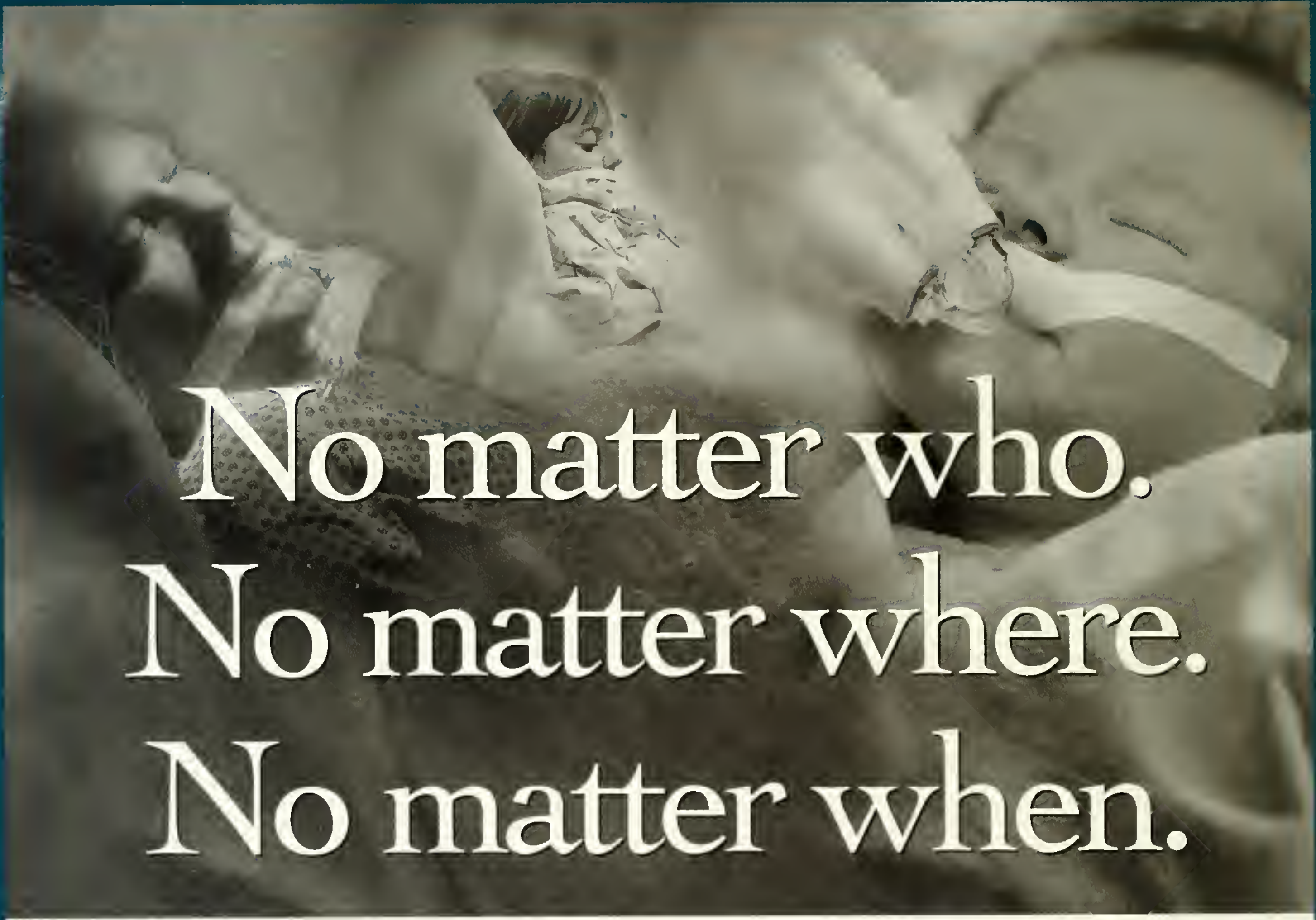
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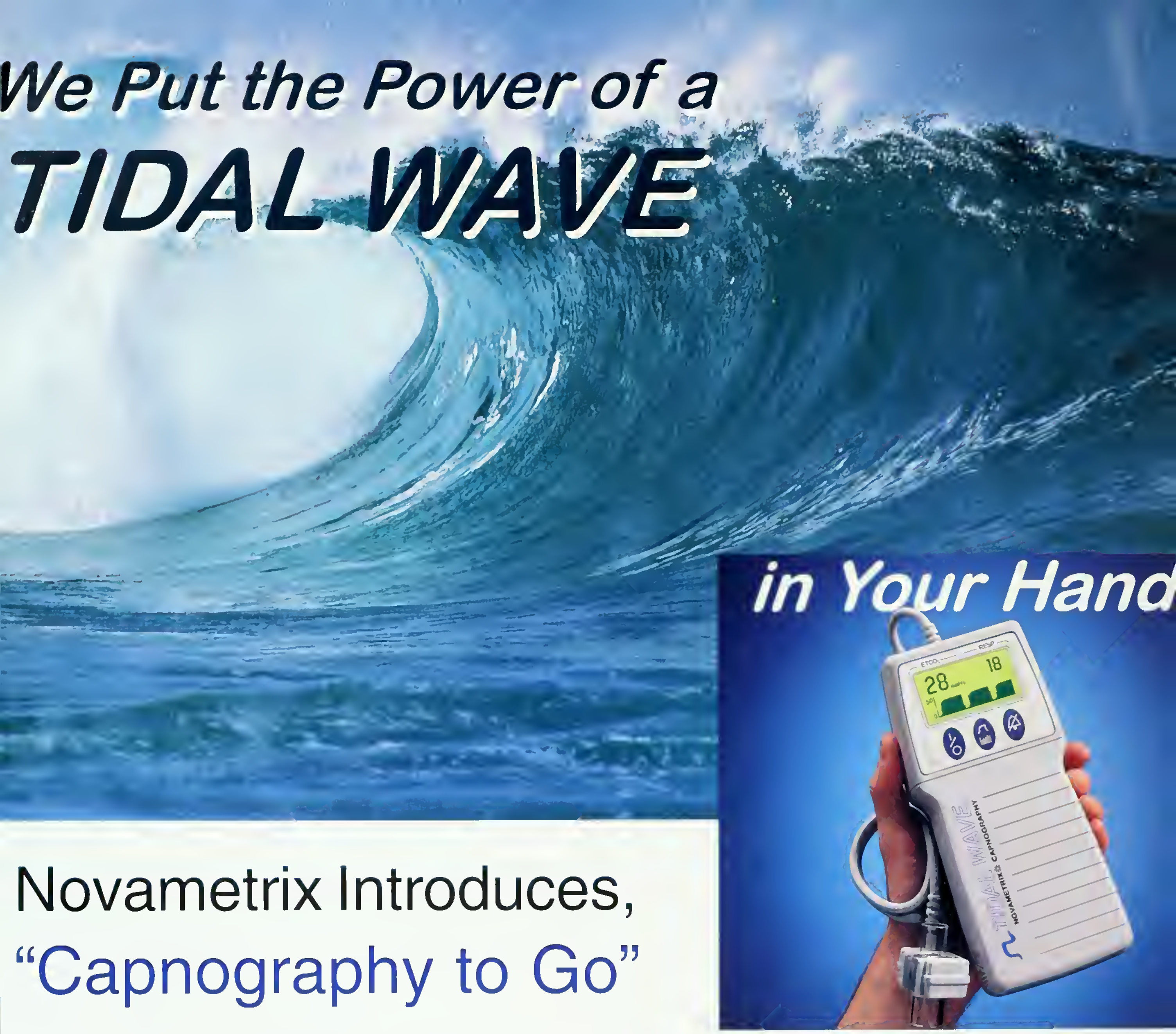

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Editorials, Commentaries, and Reviews To Note

Work of Breathing: The Debate Continues—MM Levy. Chest 1997;111(1):225-227.

Work of Breathing: The Authors Respond—RR Kirby, MJ Banner, PB Blanch. Chest 1997;111(1):228-229.

Wrong Turns in Sudden Infant Death Syndrome Research (Commentary)—AB Bergman. Pediatrics 1997;99(1):119-121.

Mucociliary Clearance in the Airways (State of the Art)—A Wanner, M Salathé, TC O'Riordan. Am J Respir Crit Care Med 1996;154:1868.

Ethical Considerations in Pulmonary Genetic Testing and Gene Therapy (Perspective)—A Kimyai-Asadi, PB Terry. Am J Respir Crit Care Med 1997;155:3-8.

Outcome of Out-of-Hospital Cardiac or Respiratory Arrest in Children—MB Schindler, D Bohn, PN Cox, BW McCrindle, A Jarvis, J Edmonds, G Barker. N Engl J Med 1996;335 (20):1473.

BACKGROUND: Among adults who have a cardiac arrest outside the hospital, the survival rate is known to be poor. However, less information is available on out-of-hospital cardiac arrest among children. This study was performed to determine the survival rate among children after out-of-hospital cardiac arrest and to identify predictors of survival. METHODS: We reviewed the records of 101 children (median age, 2 years) with apnea or no palpable pulse (or both) who presented to the emergency department at the Hospital for Sick Children in Toronto. The characteristics of the patients and the outcomes of illness were analyzed. We assessed the functional outcome of the survivors using the Pediatric Cerebral and Overall Performance Category scores. RESULTS: Overall, there was a return of vital signs in 64 of the 101 patients; 15 survived to discharge from the hospital, and 13 were alive 12 months after discharge. Factors that predicted survival to hospital discharge included a short interval between the arrest and arrival at the hospital, a palpable pulse on presentation, a short duration of resuscitation in the emergency department, and the administration of fewer doses of epinephrine in the emergency department. No patients who required more than 2 doses of epinephrine or resuscitation for longer than 20 minutes in the emergency department survived to hospital discharge. The survivors who were neurologically normal after arrest had had a respiratory arrest only and were resuscitated within 5 minutes after arrival in the emergency department. Of the 80 patients who had had a cardiac arrest, only 6 survived to hospital discharge, and all had neurologic sequelae. CONCLUSIONS: These results suggest that out-of-hospital cardiac arrest among children has a very poor prognosis, especially when efforts at resuscitation continue for longer than 20 minutes and require more than 2 doses of epinephrine.

Promoting Abstinence among Relapsed Chronic Smokers: The Effect of Telephone Support—HA Lando, PL Pirie, J Roski, PG McGovern, LA Schmid. Am J Public Health 1996;86(12):1786.

OBJECTIVES: The present study used telephone support both to sustain abstinence and to encourage renewed quit attempts in smokers who had completed an intensive smoking cessation clinic. METHODS: Subjects were hardcore smokers (n = 1,083) who had attended a multisession cessation clinic. They were then assigned randomly to receive telephone support (intervention calls 3, 9, and 21 months after the targeted cessation clinic quit date) or no further intervention. RESULTS: In the intervention condition, subjects who relapsed were significantly more likely to resume abstinence (that is, to recycle) than those in the comparison condition at follow-up (6 months: 17.8% vs 11.3%; 24 months: 25.7% vs 18.2%). Telephone support was not effective in preventing relapse, and overall differences in abstinence outcome were not significant. CONCLUSIONS: The major hypothesis of the current study—that telephone support would enhance the resumption of abstinence—received partial support. However, there was no evidence either of an overall treatment effect or of an effect in preventing relapse. Telephone outreach may be more effective in the context of self-help or other less intensive interventions.

The Long-Term Effects of a Cardiovascular Disease Prevention Trial: The Stanford Five-City Project—MA Winkleby, CB Taylor, D Jatulis, SP Fortmann. Am J Public Health 1996;86 (12):1773.

OBJECTIVES: This study examined long-term effects of a health-education intervention trial to reduce the risk of cardiovascular disease. METHODS: Surveys were conducted in California in 2 treatment and 2 control cities at baseline (1979/1980), after the 6-year intervention (1985/1986), and 3 years later at follow-up (1989/1990). Net treatment/control differences in risk-factor change were assessed for women and men 25 to 74 years of age. RESULTS: Blood pressure improvements observed in all cities from baseline to the end of the intervention were maintained during the follow-up in treatment but not control cities. Cholesterol levels continued to decline in all cities during follow-up. Smoking rates leveled out or increased slightly in treatment cities and continued to decline in control cities but did not yield significant net differences. Both coronary heart disease and all-cause mortality risk scores were maintained or continued to improve in treatment cities while leveling out or rebounding in control cities. CONCLUSIONS: These findings suggest that community-based cardiovascular disease prevention trials can have sustained effects. However, the modest net differences in risk factors suggest the need for new designs and interventions that will accelerate positive risk-factor change.

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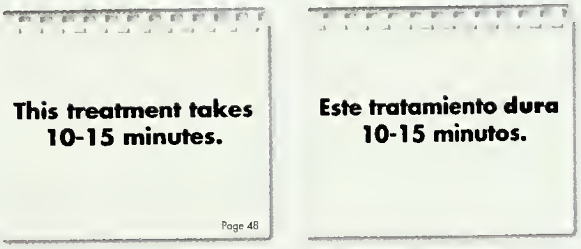

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Factors Associated with Prolongation of Transport Times of Emergency Pediatric Patients Requiring Transfer to a Tertiary Care Center—FC Beddingfield, HG Garrison, JE Manning, RJ Lewis. Pediatric Emergency Care 1996;12(6):416.

PURPOSE: The purpose of this study was to determine factors associated with longer times to transport of emergency pediatric patients requiring tertiary care. DESIGN: Retrospective case series. SETTING: Emergency pediatric transport service. PARTICIPANTS: Infants and children transported by the transport service at the University of North Carolina Hospitals at Chapel Hill from January 1, 1988, to December 31, 1990. MAIN MEASUREMENTS: The time-to-request, the time from patient arrival at the referring hospital to the time when the request for transfer was received, and the ground time, defined as the time between the transport team's arrival at the referring hospital and their departure, were recorded for each transported patient. RESULTS: Three hundred consecutive children 0 to 16 years (61% male) were transferred. Time-to-request was shorter for trauma patients (median 62 min, quartiles 29 and 153 min) than for medical patients (median 172 min, quartiles 83 and 508 min) ($p = 0.0001$). Infants, children, and adolescents had similar times-to-request of 147 minutes, 129 minutes, and 128 minutes, respectively ($p = 0.91$). Increased ground times were associated with diagnosis category (median of 40 min for medical patients vs 29 min for trauma patients) ($p = 0.0001$), with younger age (median of 46 min for infants, 35 min for children, and 28 min for adolescents) ($p = 0.0001$), and with the performance of major procedures (median of 35 min if no procedures were performed, 38 min if 1 procedure was performed, and 54 min if 2 procedures were performed) ($p = 0.039$). After the transport team arrived, 13% (40/300) of patients required at least 1 major procedure prior to transport. CONCLUSIONS: Increased time-to-request for patients with medical diagnoses, increased ground times for younger patients and patients with medical diagnoses, and failure to perform necessary procedures contribute to a prolongation of the time-to-transport of emergency pediatric patients. The magnitude of the impact of these longer transport times on outcome is unknown.

U.S. Emergency Department Costs: No Emergency—PH Tyrance Jr, DU Himmelstein, S Woolhandler. Am J Public Health 1996;86(11):1527.

BACKGROUND: Many perceive emergency department (ED) overuse as an important cause of high medical care costs in the United States. Managed care plans and politicians have seen constraints on ED use as an important element of cost control. METHODS: We measured ED-associated and other medical care costs, using the recently released 1987 National Medical Expenditure Survey of approximately 35,000 persons in 14,000 households representative of the U.S. civilian, noninstitutionalized population. RESULTS: In 1987, total ED expenditures were $8.9 billion, or 1.9% of national health expenditures. People with health insurance represented 86% of the population and accounted for 88% of ED spending. The uninsured paid 47% of ED costs themselves; free care covered only 10%. For the uninsured, the cost of hospitalization initiated by ED visits totaled $3.3 billion, including $1.1 billion in free care. Whites accounted for 75% of total ED costs. The ED costs of poor and near-poor individuals accounted for only 0.47% of national health costs. CONCLUSIONS: ED use accounts for a small share of U.S. medical care costs, and cost shifting to the insured to cover free ED care for the uninsured is modest. Constraining ED use cannot generate substantial cost savings but may penalize minorities and the poor, who receive much of their outpatient care in EDs.

Improved Survival with an Implanted Defibrillator in Patients with Coronary Disease at High Risk for Ventricular Arrhythmia—AJ Moss, WJ Hall, DS Cannom, JP Daubert, SL Higgins, et al, for the Multicenter Automatic Defibrillator Implantation Trial Investigators. N Engl J Med 1996;335(26):1933.

BACKGROUND: Unsustained ventricular tachycardia in patients with previous myocardial infarction and left ventricular dysfunction is associated with a 2-year mortality rate of about 30%. We studied whether prophylactic therapy with an implanted cardioverter-defibrillator, as compared with conventional medical therapy, would improve survival in this high-risk group of patients. METHODS: Over the course of 5 years, 196 patients in New York Heart Association functional class I, II, or III with prior myocardial infarction; a left ventricular ejection fraction ≤ 0.35; a documented episode of asymptomatic unsustained ventricular tachycardia; and inducible, nonsuppressible ventricular tachyarrhythmia on electrophysiologic

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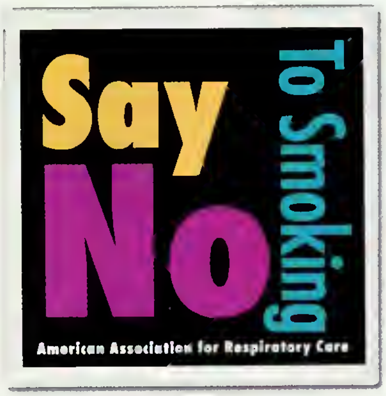

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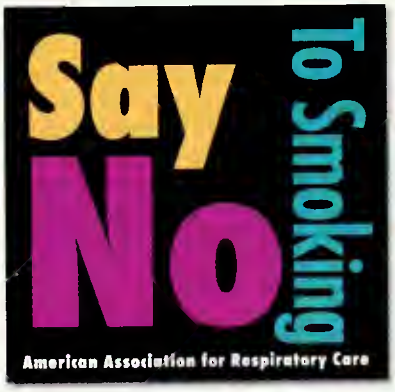

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study were randomly assigned to receive an implanted defibrillator ($n = 95$) or conventional medical therapy ($n = 101$). We used a 2-sided sequential design with death from any cause as the end point. RESULTS: The baseline characteristics of the 2 treatment groups were similar. During an average follow-up of 27 months, there were 15 deaths in the defibrillator group (11 from cardiac causes) and 39 deaths in the conventional-therapy group (27 from cardiac causes) (hazard ratio for overall mortality, 0.46; 95% confidence interval, 0.26 to 0.82; $p = 0.009$). There was no evidence that amiodarone, β-blockers, or any other antiarrhythmic therapy had a significant influence on the observed hazard ratio. CONCLUSIONS: In patients with a prior myocardial infarction who are at high risk for ventricular tachyarrhythmia, prophylactic therapy with an implanted defibrillator leads to improved survival as compared with conventional medical therapy.

The Safety of Transdermal Nicotine as an Aid to Smoking Cessation in Patients with Cardiac Disease—AM Joseph, SM Norman, LH Ferry, AV Prochazka, EC Westman, BG Steele, et al. N Engl J Med 1996;335:1792.

BACKGROUND: Transdermal nicotine therapy is widely used to aid smoking cessation, but there is uncertainty about its safety in patients with cardiac disease. METHODS: In a randomized, double-blind, placebo-controlled trial at 10 Veterans Affairs medical centers, we randomly assigned 584 outpatients (of whom 576 were men) with at least 1 diagnosis of cardiovascular disease to a 10-week course of transdermal nicotine or placebo as an aid to smoking cessation. The subjects were monitored for a total of 14 weeks for the primary end points of the study (death, myocardial infarction, cardiac arrest, and admission to the hospital due to increased severity of angina, arrhythmia, or congestive heart failure); the secondary end points (admission to the hospital for other reasons and outpatient visits necessitated by increased severity of heart disease); any side effects of therapy; and abstinence from smoking. RESULTS: There were 48 primary and 78 secondary end points noted in a total of 95 subjects. At least 1 of the primary end points was reached by 5.4% of the subjects in the nicotine group and 7.9% of the subjects in the placebo group (difference, 2.5%; 95% confidence interval, −1.6 to 6.5%; $p = 0.23$). In the nicotine group, 11.9% of the subjects had at least 1 of the secondary end points, as compared with 9.7% in the placebo group (difference, 2.2%; 95% confidence interval, −2.2 to 7.4%; $p = 0.37$). After 14 weeks the rate of abstinence from smoking was 21% in the nicotine group, as compared with 9% in the placebo group ($p = 0.001$), but after 24 weeks the abstinence rates were not significantly different (14% vs 11%, $p = 0.67$). CONCLUSIONS: Transdermal nicotine does not cause a significant increase in cardiovascular events in high-risk outpatients with cardiac disease. However, the efficacy of transdermal nicotine as an aid to smoking cessation in such patients is limited and may not be sustained over time.

Cardiopulmonary Resuscitation: What Cost To Cheat Death?—KH Lee, DC Angus, NS Abramson. Crit Care Med 1996;24(12):2046.

OBJECTIVES: To review the various outcomes from cardiopulmonary resuscitation (CPR), the factors that influence these outcomes, the costs associated with CPR, and the application of cost-analyses to CPR. DATA SOURCES: Data used to prepare this article were drawn from published articles and work in progress. STUDY SELECTION: Articles were selected for their relevance to the subjects of CPR and cost-analysis by MEDLINE keyword search. DATA EXTRACTION: The authors extracted all applicable data from the English literature. DATA SYNTHESIS: Cost-analysis studies of CPR programs are limited by the high variation in resources consumed and attribution of cost to these resources. Furthermore, cost projections have not been adjusted to reflect patient-dependent variation in outcome. Variation in the patient's underlying condition, presenting cardiac rhythm, time to provision of definitive CPR, and effective perfusion all influence final outcome and, consequently, influence the cost-effectiveness of CPR programs. Based on cost data from previous studies, preliminary estimates of the cost-effectiveness of CPR programs for all 6-month survivors of a large international multicenter collaborative trial are $406,605/life saved (range $344,314 to $966,759), and $225,892/quality-adjusted-life-year (range $191,286 to $537,088). CONCLUSIONS: Reported outcome from CPR has varied from reasonable rates of good recovery, including return to full employment to 100% mortality. Appropriate CPR is encouraged, but continued widespread application appears extremely expensive.

Evaluation of Tuberculosis Control Policies Using Computer Simulation—TF Brewer, SJ Heymann, GA Colditz, ME Wilson, K Auerbach, D Kane, HV Fineberg. JAMA 1996;276(23):1898.

OBJECTIVE: To develop more effective methods to assess tuberculosis (TB) control strategies so we can meet national goals for the elimination of TB in the United States. DESIGN: Using a semi-Markov model that divided the U.S. population into 3 age groups and 18 clinical states based on disease status and risk for TB and human immunodeficiency virus (HIV) infection, we measured the effects of 5 changes in TB policy, introduced singly and in combination: (1) increased coverage and (2) improved efficacy of preventive therapy, (3) increased coverage and (4) improved efficacy of treatment, and (5) introduction of BCG vaccination. RESULTS: A BCG vaccination program that reached 10% of eligible children and 1% of eligible adults each year would produce a 17% reduction in cases and an 11% decline in deaths over 10 years. Preventive therapy programs among the general population would have little effect on the number of TB cases, but a program targeting HIV-infected patients would reduce HIV-associated TB cases and deaths 14% to 20%. A 10% improvement in the coverage and efficacy of both preventive therapy and treatment, coupled with the BCG vaccination program, would lead to a 47% decline in TB cases and a 50% decline in TB deaths relative to baseline over 10 years. CONCLUSIONS: Improvements in treatment coverage or effectiveness alone are unlikely to reach established national goals for the elimination of TB. These goals can be achieved through a combination of improvements in current programs with targeted preventive therapy and BCG vaccination programs. *See the related editorial:* ***Sharpen Available Tools for Tuberculosis Control, But New Tools Needed for Elimination****—B Miller, KG Castro. JAMA 1996;276(23):1916-1917.*

Gradual Reduction of Endotracheal Tube Diameter during Mechanical Ventilation via Different Humidification Devices—MC Villafane, G Cinnella, F Lofaso, D Isabey, A Harf, F Lemaire, L Brochard. Anesthesiology 1996;85(6):1341.

BACKGROUND: Limited data suggest that increased resistance to flow within endotracheal tubes (ETT) may occur in patients whose lungs are mechanically ventilated for more than 48 hours, especially when airway humidification is inadequate. This could lead to sudden ETT obstruction or induce excessive loading during spontaneous breathing. METHODS: Twenty-three such patients were randomly assigned to 3 types of airway humidifier based on 3 different working principles: a Fisher & Paykel hot water system ($n = 7$), a Pall BB2215 heat and moisture exchanger (HME) hydrophobic filter ($n = 8$), and a Dar Hygrobac 35254111 HME hygroscopic filter ($n = 8$). The decrease in internal pressure along the ETT and the flow rate were measured in each patient every 2 days. An "effective inner diameter" was derived from those measurements and allowed the inner ETT configuration to be monitored. RESULTS: On the first day of intubation, the mean diameter was similar in the 3 groups, and was slightly smaller than the in vitro diameter (mean ± SD: 7.6 ± 0.6 mm for Fisher & Paykel, 7.7 ± 0.4 for Pall, and 7.5 ± 0.4 for Dar). The mean diameter tended to decrease from day to day. At the end of the study, the overall reduction in mean diameter was significantly greater with the hydrophobic HME (Pall) than with the 2 other systems (Pall: −6.5 ± 4% vs −2.5 ± 2.5% for Dar and −1.5 ± 3% for Fisher & Paykel; $p < 0.01$ with analysis of variance). The same was true of the mean reduction in effective inner ETT diameter expressed per day of ventilation (−1.6 ± 1.5% per day for Pall vs −0.5 ± 0.4% for Dar and −0.2 ± 0.4% for Fisher & Paykel; $p < 0.01$). In 4

March 1997
How to Resuscitate a Lizard
Lawrence Bowman, RRT
1 Scoop lizard from pool
2 Shake out lizard
3 Apply on and off pressure to chest
4 Apply mouth to mouth resuscitation
20 Spring Begins
17 St. Patrick's Day
28 Good Friday
23 Palm Sunday
Easter 30
February 1997
"The doctors told me when you start baseball, you had an operation. I told Mom I you aren't going to be able wouldn't need to run that baseball, I'll just hit them out of the park. Then I'll be able to walk."
Edward J. McGrath, Jr., from "An Exceptional View of Life"
14 Valentine's Day
22 Washington's Birthday
12 Ash Wednesday Lincoln's Birthday
17 President's Day
Working in home care has its unique problems
August 1997
...ver encountered it.
...re temporary setbacks!
Dottie Walters

$3

Item R46

($5 nonmembers)

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Early deadline: March 17, 1997

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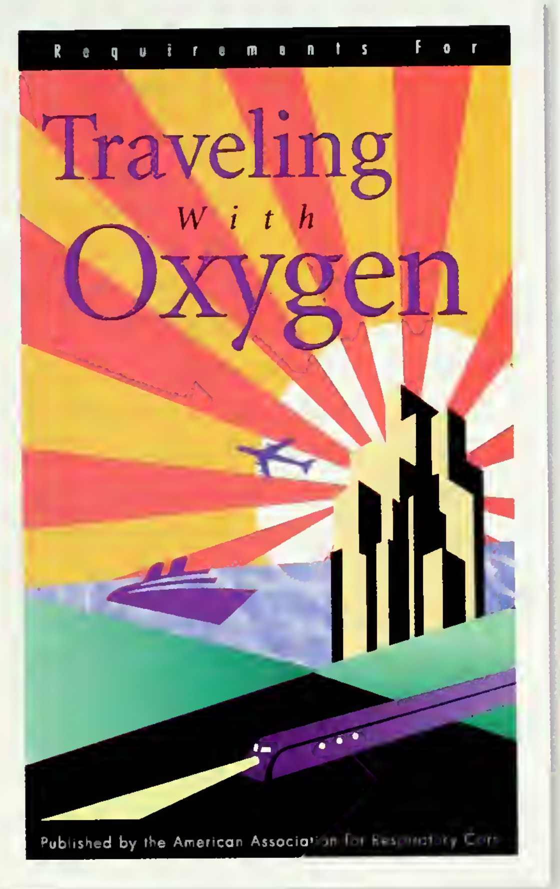

Requirements For

Traveling With Oxygen

Published by the American Association for Respiratory Care

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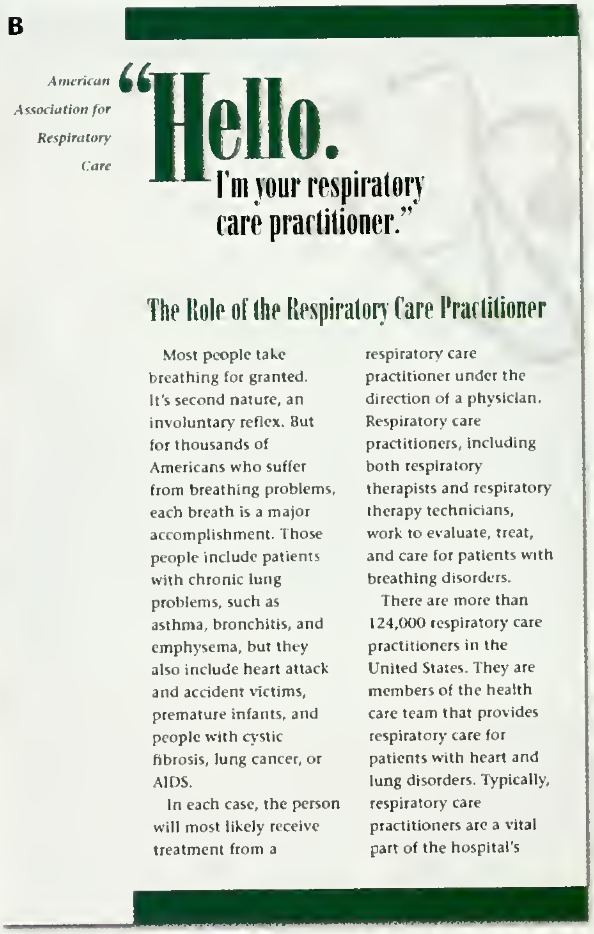


American Association for Respiratory Care

"Hello. I'm your respiratory care practitioner."

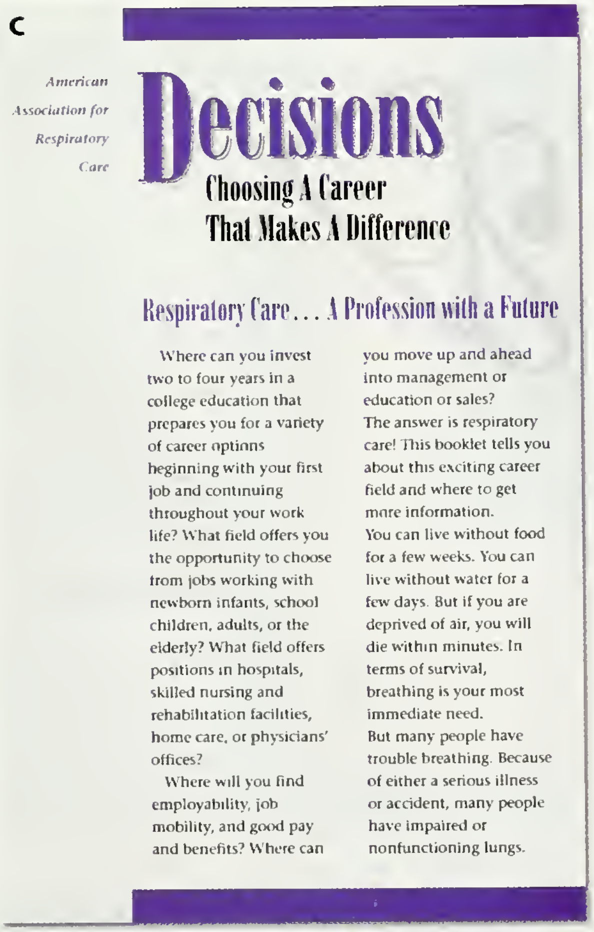
The Role of the Respiratory Care Practitioner

Most people take breathing for granted. It's second nature, an involuntary reflex. But for thousands of Americans who suffer from breathing problems, each breath is a major accomplishment. Those people include patients with chronic lung problems, such as asthma, bronchitis, and emphysema, but they also include heart attack and accident victims, premature infants, and people with cystic fibrosis, lung cancer, or AIDS.

In each case, the person will most likely receive treatment from a respiratory care practitioner under the direction of a physician. Respiratory care practitioners, including both respiratory therapists and respiratory therapy technicians, work to evaluate, treat, and care for patients with breathing disorders.

There are more than 124,000 respiratory care practitioners in the United States. They are members of the health care team that provides respiratory care for patients with heart and lung disorders. Typically, respiratory care practitioners are a vital part of the hospital's

C



American Association for Respiratory Care

Decisions

Choosing A Career That Makes A Difference

Respiratory Care . . . A Profession with a Future

Where can you invest two to four years in a college education that prepares you for a variety of career options beginning with your first job and continuing throughout your work life? What field offers you the opportunity to choose from jobs working with newborn infants, school children, adults, or the elderly? What field offers positions in hospitals, skilled nursing and rehabilitation facilities, home care, or physicians' offices?

Where will you find employability, job mobility, and good pay and benefits? Where can you move up and ahead into management or education or sales? The answer is respiratory care! This booklet tells you about this exciting career field and where to get more information.

You can live without food for a few weeks. You can live without water for a few days. But if you are deprived of air, you will die within minutes. In terms of survival, breathing is your most immediate need.

But many people have trouble breathing. Because of either a serious illness or accident, many people have impaired or nonfunctioning lungs.

Requirements For Traveling With Oxygen

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B This brochure, with a new look, is packed with information that tells your patients and others in the community exactly who you are and what you do. Package of 50.

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"Decisions: Choosing A Career That Makes A Difference" NEW

C Inform people searching for a career about opportunities and benefits available in the respiratory care field. This brochure also looks at the qualifications and skills needed to become an RCP. Package of 50.

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With the AARC Richard Branson Doesn't Fly By the Seat of His Pants

As a professional Registered Respiratory Therapist, I look to the American Association for Respiratory Care for guidance, advice, and information. Without this foundation on which I practice and depend, I would just be 'flying by the seat of my pants.'

Although I joined the AARC as a student in 1978, it was not until 1984 when I attended my first meeting in Phoenix that I really knew my place in the organization. It was there in a lightning-quick half hour for RESPIRATORY CARE first-time authors that I met some of the most influential people—Phil Kittredge, Dean Hess, Rob Chatburn, Chris Maxwell—in the field. I'm proud to say many of those I met that day have remained my lifelong friends. I look to the AARC for invaluable resources, including *AARC Times*, Clinical Practice Guidelines, videos, and patient education sheets. However, in the legion of throwaways and want-to-be journals, the one, true voice of the professional respiratory care practitioner is RESPIRATORY CARE; and, I am honored to be part of its editorial board and proud to be a member of the AARC—an organization that helps make me who I am. Why not let it do the same for you?

Richard Branson, RRT
Cincinnati, Ohio

Forrest Bird, MD, and Richard Branson, RRT

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American Association for Respiratory Care

Membership Application

Please read the eligibility requirements for each of the classifications in the right-hand column, then complete the applicable section. All information requested below must be provided, except where indicated as optional. See other side for more information and fee schedule. Please sign and date application on reverse side and type or print clearly. Processing of application takes approximately 15 days.

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    ☐ Foreign
    ☐ Physician
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☐ Special
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Phone No. (______) ____________________

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☐ Instructor/Educator
☐ Supervisor
☐ Staff Therapist
☐ Staff Technician
☐ Rehabilitation/Home Care
☐ Medical Director
☐ Sales
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Type of Business

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☐ Home Health Agency
☐ Educational Institution
☐ Manufacturer or supplier
☐ Other, specify ____________________

Date of Birth (optional) __________ Sex (optional) __________

U.S. Citizen? _____ Yes _____ No

Have you ever been a member of the AARC? ____________________

If so, when? From ______________ to ______________

Preferred mailing address: ☐ Home ☐ Business

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An individual is eligible if he/she lives in the U S. or its territories or was an Active Member prior to moving outside its borders or territories, and meets ONE of the following criteria: (1) is legally credentialed as a respiratory care professional if employed in a state that mandates such, OR (2) is a graduate of an accredited educational program in respiratory care, OR (3) holds a credential issued by the NBRC. An individual who is an AARC Active Member in good standing on December 8, 1994, will continue as such provided his/her membership remains in good standing

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SPECIAL NOTICE — Student Members do not receive Continuing Respiratory Care Education (CRCE) transcripts. Upon completion of your respiratory care education, continuing education credits may be pursued upon your reclassification to Active or Associate Member.

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City ____________________

State __________ Zip __ __ __ __ __ - __ __ __ __

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Length of program

☐ 1 year ☐ 4 years
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Expected Date of Graduation (REQUIRED INFORMATION)

Month _______ Year _______

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Demographic Questions

We request that you answer these questions in order to help us design services and programs to meet your needs.

Check the Highest Degree Earned

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- ☐ RC Graduate Technician
- ☐ Associate Degree
- ☐ Bachelor's Degree
- ☐ Master's Degree
- ☐ Doctorate Degree

Number of Years in Respiratory Care

- ☐ 0-2 years
- ☐ 3-5 years
- ☐ 6-10 years
- ☐ 11-15 Years
- ☐ 16 years or more

Job Status

- ☐ Full Time
- ☐ Part Time

Credentials

- ☐ RRT
- ☐ CRTT
- ☐ Physician
- ☐ CRNA
- ☐ RN
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- ☐ Less than $10,000
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- ☐ $20,001-$30,000
- ☐ $30,001-$40,000
- ☐ $40,000 or more

PLEASE SIGN

I hereby apply for membership in the American Association for Respiratory Care and have enclosed my dues. If approved for membership in the AARC, I will abide by its bylaws and professional code of ethics. I authorize investigation of all statements contained herein and understand that misrepresentations or omissions of facts called for is cause for rejection or expulsion.

A yearly subscription to RESPIRATORY CARE journal and *AARC Times* magazine includes an allocation of $6.50 from my dues for each of these publications.

NOTE: Contributions or gifts to the AARC are not tax deductible as charitable contributions for income tax purposes. However, they may be tax deductible as ordinary and necessary business expenses subject to restrictions imposed as a result of association lobbying activities. The AARC estimates that the nondeductible portion of your dues — the portion which is allocable to lobbying — is 26%.

Signature ______________________________

Date ______________________________

Membership Fees

Payment must accompany your application to the AARC. Fees are for 12 months. (NOTE: Renewal fees are $65.00 Active, Associate-Industrial or Associate-Physician, or Special status; $80.00 for Associate-Foreign status; and $35.00 for Student status).

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☐ Associate (Industrial or Physician)	$77.50
☐ Associate (Foreign)	$92.50
☐ Special	$77.50
☐ Student	$35.00
TOTAL	$__________

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Established to recognize the specialty areas of respiratory care, these sections publish a newsletter four times a year that focuses on issues of specific concern to that specialty. The sections also design the specialty programming at the national AARC meetings.

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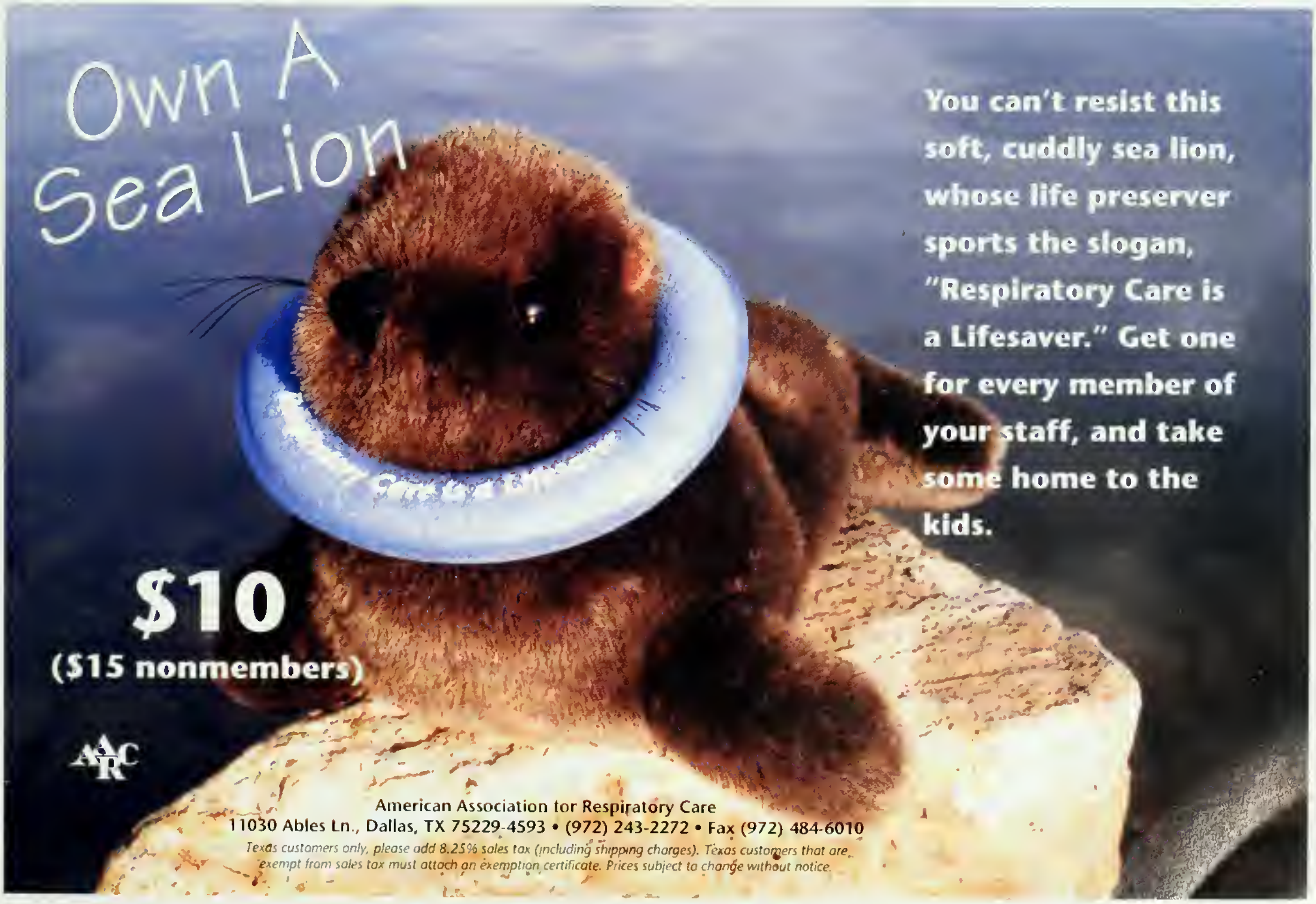
patients, the ETT became obstructed and emergency repeated tracheal intubation was required. The Pall HME and the Fisher & Paykel system were being used in 3 and 1 patient, respectively. Before obstruction, the reduction in ETT diameter was significantly greater for these 4 patients than for the remaining 23 patients (7.8 ± 1.4% vs 3.1 ± 4.1%; $p < 0.01$). CONCLUSIONS: During prolonged mechanical ventilation, significant alterations in inner ETT configuration occur frequently and are influenced by the type of humidification device used. In vivo monitoring of ETT mechanical properties might be clinically useful.

Early Tracheal Extubation after Coronary Artery Bypass Graft Surgery Reduces Costs and Improves Resource Use: A Prospective, Randomized, Controlled Trial—DCH Cheng, J Karski, C Peniston, G Raveendran, B Asokumar, J Carroll, T David, A Sandler. Anesthesiology 1996;85(6):1300.

BACKGROUND: Economics has caused the trend of early tracheal extubation after cardiac surgery, yet no prospective randomized study has directly validated that early tracheal extubation anesthetic management decreases costs when compared with late extubation after cardiac surgery. METHODS: This prospective, randomized, controlled clinical trial was designed to evaluate the cost savings of early (1-6 hours) versus late tracheal extubation (12-22 hours) in patients after coronary artery bypass graft (CABG) surgery. The total cost for the services provided for each patient was determined for both the early and late groups from hospital admission to discharge home. All costs applicable to each of the services were classified into direct variables, direct fixed costs, and overhead (an indirect cost). Physician fees and heart catheterization costs were included. The total service cost was the sum of unit workload and overhead costs. RESULTS: One hundred patients having elective CABG who were younger than 75 years were studied. Including all complications, early extubation (n = 50) significantly reduced cardiovascular intensive care unit (CVICU) costs by 53% ($p < 0.026$) and the total CABG surgery cost by 25% ($p < 0.019$) when compared with late extubation (n = 50). Forty-one patients (82%) in each group were tracheally extubated within the defined period. In the early extubation group, the actual departmental cost savings in CVICU nursing and supplies was 23% ($p < 0.005$), in ward nursing and supplies was 11% ($p < 0.05$), and in respiratory therapy was 12% ($p < 0.05$). The total cost savings per patient having CABG was 9% ($p < 0.001$). Further cost savings using discharge criteria were 51% for CVICU nursing and supplies ($p < 0.001$), 9% for ward nursing and supplies ($p < 0.05$), and 29% for respiratory therapy ($p < 0.001$), for a total cost savings per patient of 13% ($p < 0.001$). Early extubation also reduced elective case cancellations ($p < 0.002$) without any increase in the number of postoperative complications and readmissions. CONCLUSIONS: Early tracheal extubation anesthetic management reduces total costs per CABG surgery by 25%, predominantly in nursing and in CVICU costs. Early extubation reduces CVICU and hospital length of stay but does not increase the rate or costs of complications when compared with patients in the late extubation group. It shifts the high CVICU costs to the lower ward costs. Early extubation also improves resource use after cardiac surgery when compared with late extubation.


Own A Sea Lion

You can't resist this soft, cuddly sea lion, whose life preserver sports the slogan, "Respiratory Care is a Lifesaver." Get one for every member of your staff, and take some home to the kids.

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Item R8. Package of 25.

$7.50

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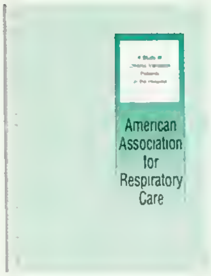

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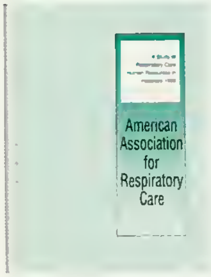

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Noninvasive Estimate of Work of Breathing Due To the Endotracheal Tube—L Heyer, B Louis, D Isabey, F Lofaso, L Brochard, JJ Fredberg, A Harf. Anesthesiology 1996;85(6):1324.

BACKGROUND: Although evidence suggests that secretions lining the inner wall of the endotracheal tube (ETT) often reduce its cross-sectional area, no data are available on the work of breathing as affected by the ETT. A noninvasive method is proposed for estimating the additional work of breathing necessitated by the ETT in patients whose lungs are mechanically ventilated. This method (the acoustic-Blasius method) involves (1) determining the inner geometry of the ETT using the acoustic reflection method and (2) using these geometric data to solve the Blasius equation that characterizes the ETT pressure drop-flow relation. METHODS: To evaluate the acoustic-Blasius method in vivo, the authors computed the work of breathing due to the ETT in 4 healthy persons breathing through an ETT connected to a pressure-support device and in 5 tracheally intubated patients receiving mechanical assistance in the pressure-support mode. For the tracheally intubated patients, the reference value was the work calculated from the ETT pressure drop measured between the 2 ends of the ETT using a pressure catheter. RESULTS: In the healthy participants and the tracheally intubated patients, there was close agreement between inspiratory work per cycle values estimated by directly measuring the ETT pressure drop and calculated using the acoustic-Blasius method: The difference was consistently less than 0.08 joules (< 10% of the reference value). CONCLUSIONS: The data show that the acoustic-Blasius method allows noninvasive quantification of the ETT-related work of breathing in situ.

Are Bedding and Rebreathing Suffocation a Cause of SIDS? (Commentary)—WG Guntheroth, PS Spiers. Pediatr Pulmonol 1996;22:335.

Suffocation by bedclothes became a popular diagnosis in the 1940s but gradually became replaced with the diagnostic label of Sudden Infant Death Syndrome (SIDS). In 1991 a paper purported that, instead of SIDS, pillows filled with polystyrene beads had caused death by rebreathing suffocation; this conclusion was reached on the basis of experiments with anesthetized rabbits breathing through a doll's head that was placed face down on the pillow. Because of the anesthesia, rabbits could not change their face down position. The doll's nares could not collapse, which would have resulted in rapid death due to conventional suffocation. The rabbits required up to 3 hours or more to die of hypercarbia and hypoxia. Studies in normal infants revealed that they turned from the face-down position after only 2 minutes. (The only infant who retained CO_2 soon died of a fatal neurologic disorder, with central hypoventilation). Using the rabbit/doll's head and mechanical models, a wide range of bedding was indicted, including cushions, sheepskins, pillows, comforters, foam mattresses, and even simple blankets and sheets as potentially causing fatal rebreathing. Except for the use of pillows in general, as well as mattresses filled with kapok and bark, there has been no epidemiologic support for these indictments. Although normal infants are unlikely to succumb to rebreathing suffocation, infants with blunted ventilatory responsiveness and delayed arousal due to prior hypoxia were hypothesized to be at increased risk. Support for this concept was found in the pathology of the brain stem in victims of SIDS that was attributed to prior hypoxic injury. In infants who survived prolonged apnea, < 20% have demonstrated a diminished ventilatory responsiveness to hypercarbia, but, more significantly, none had an absent response. Arousal to hypercarbia, an abnormality which is crucial to the hypothesis of rebreathing suffocation, is regularly present in normal subjects, but the threshold is higher in near-SIDS infants; however, no instances of failure to arouse have been reported

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in near-SIDS. If the infant is placed on his or her back or side, the issue of bedding could become moot; unfortunately, a sizable percentage of infants are still being placed prone for sleep. Instead of confusing parents with an ever-expanding list of "dangerous bedding," the message "Back to Sleep" should be emphasized.

The Influence of Sleeping Position on Functional Residual Capacity and Effective Pulmonary Blood Flow in Healthy Neonates—NR Aiton, GF Fox, J Alexander, DM Ingram, AD Milner. Pediatr Pulmonol 1996;22:342.

Variation in body position has been shown to affect respiratory function in adults and neonates with and without respiratory illness. At present it remains unclear why respiratory function should be affected by different body positions. We hypothesized that the effect of body weight on the relatively compliant chest wall of the newborn infant in the prone position would cause a reduction in functional residual capacity (FRC) and a compensatory improvement in ventilation/perfusion matching as measured by effective pulmonary blood flow. To evaluate this, a paired crossover study was performed on 12 normal newborn infants. The inert gas (argon) rebreathing method adapted for neonates was used to measure FRC. Simultaneously effective pulmonary blood flow ($Q_{P_{eff}}$) was determined using Freon 22 and a mass spectrometer with computerized analysis. The babies were studied in 3 different positions in random order: prone, supine, and right lateral decubitus. The means (95% confidence intervals) of the 3 groups for FRC were 23.8 (19.2 to 28.4), 23.8 (20.2 to 27.5), and 24.3 (19.5 to 29.2) mL/kg, respectively ($p = 0.59$), and for $Q_{P_{eff}}$ were 104 (91 to 116), 108 (95 to 122), 109 (97 to 122) mL/kg-min, respectively ($p = 0.11$). Thus no significant differences were demonstrated. In 9 of the babies, a repeat supine measurement was taken at the end of the study to assess repeatability of the method. In these 9 babies alone the results were 22.7 (19.1 to 26.3) and 22.1 (18.6 to 25.6) mL/kg for FRC, and 102 (89 to 116) and 98 (90 to 107) mL/kg-min for $Q_{P_{eff}}$. The coefficients of repeatability were 4.7 mL/kg for FRC (21%) and 30 mL/kg-min for QP_{eff} (30%). *See the related editorial: **How Should Sleeping Babies Lie?**—JG Brooks. Pediatr Pulmonol 1996;22:333-334.*

Lung Function Tests in Patients With Idiopathic Pulmonary Fibrosis: Are They Helpful for Predicting Outcome?—R Erbes, T Schaberg, R Loddenkemper. Chest 1997;111(1):51.

OBJECTIVE: Idiopathic pulmonary fibrosis (IPF) varies widely in its course. To evaluate predictive parameters at presentation to the hospital, we investigated 99 patients with IPF (47 women), focusing on extensive lung function tests. METHODS: Standard tests of lung volumes, arterial oxygen tension, and gas exchange at rest and during bicycle exercise were performed. Survival rates in relation to functional parameters were calculated using the actuarial method. Differences in survival proportions were summarized as hazard ratios, and significance levels were determined by log-rank test. RESULTS: At presentation, most patients showed a reduced total lung capacity (TLC) of 79.2 ± 21.1%, an arterial oxygen tension (P_{aO_2}) considered pathologic in 63%, when related to age, a significant decrease of P_{aO_2} with 11.8 ± 12.1 mm Hg and an increase of the alveolar-arterial oxygen pressure difference with 46.4 ± 16.4 (12.2 to 76.8) mm Hg during bicycle exercise. Diminished survival was associated with an age older than 50 years, a reduced value to more than 2 SDs below the predicted values of both, TLC alone, or in combination with a reduced vital capacity. Factors not influencing survival were gender, parameters of gas exchange at rest, and P_{aO_2} at rest and during bicycle exercise. CONCLUSIONS: We conclude that standard lung function tests make it possible to assess the prognosis of patients with IPF, while extensive tests like gas exchange measurements at rest and during bicycle exercise do not contribute additional information to make the prognostic estimations more precise. *See the related editorial: **Pulmonary Function Tests and Idiopathic Pulmonary Fibrosis: Simple May Be Better**—SH Kirtland, RH Winterbauer. Chest 1997;111(1):7-8.*

The D_{CO_2} Measured by Gastric Tonometry Predicts Survival in Children Receiving Extracorporeal Life Support: Comparison with Other Hemodynamic and Biochemical Information—T Duke, W Butt, M South, F Shann, and the Royal Children's Hospital ECMO Nursing Team. Chest 1997;111(1):174.

OBJECTIVE: To assess the role of gastric tonometry in monitoring children receiving extracorporeal life support (ECLS) and to determine if D_{CO_2} or pHi in the weaning phase of ECLS predicts survival. DESIGN: A prospective study of consecutive patients treated with ECLS. SETTING: A tertiary pediatric ICU that is the ECLS referral center for Australia. PATIENTS: Twenty consecutive children receiving ECLS for cardiovascular or respiratory failure. INTERVENTIONS: All children were monitored throughout their ECLS course using a tonometer inserted into the stomach via the orogastric route. The P_{CO_2} in the tonometer balloon was measured every 4 to 6 hours and the pHi was calculated using the Henderson-Hasselbalch equation. The D_{CO_2}, which is the difference between P_{CO_2} in tonometer saline solution and arterial blood, was calculated. We compared the ability of pHi, D_{CO_2}, heart rate, mean arterial pressure, arterial pH, base deficit, and blood lactate to predict death or survival during the weaning phase. Measurements were taken on the lowest level of support, which for veno-arterial extracorporeal membrane oxygenation and ventricular assist device was defined as the lowest ECLS pump flows, and on veno-venous extracorporeal membrane oxygenation was defined as the time of lowest ECLS gas flow. Predictive power was assessed using the receivor operating characteristic (ROC) analysis on the data collected at these times. RESULTS: In the weaning phase of ECLS, the pHi was significantly lower in children who died (pHi = 7.21; 95% confidence intervals, 7.14 to 7.28) than in those who survived (pHi = 7.38; 95% confidence intervals, 7.28 to 7.47). The D_{CO_2} was significantly higher in children who died (23.6 mm Hg; 95% confidence intervals, 14.3 to 33.1) compared with survivors (4.7 mm Hg; 95% confidence intervals, −0.78 to 10.1). The area under the ROC curve was 0.95 for D_{CO_2} (and 0.88 for pHi). pHi and D_{CO_2} predicted survival better than base deficit (area under ROC curve, 0.82), blood lactate level (0.29), arterial pH (0.65), heart rate (0.62), and mean arterial pressure (0.74). CONCLUSIONS: D_{CO_2} is a clinically meaningful measurement in children receiving ECLS. A high D_{CO_2} was a good predictor of death in this series. Gastric tonometry may provide a useful measure of the adequacy of regional perfusion and oxygenation in this group of patients. *See the related editorial: **Monitoring Tissue Oxygenation: The Search for the Grail.** DR Dantzker. Chest 1997; 111(1):12-14.*

Reduction of Environmental Tobacco Smoke Exposure in Asthmatic Children: A 2-Year Follow-up—DR Wahlgren, MF Hovell, SB Meltzer, CR Hofstetter, JM Zakarian. Chest 1997;111 (1):81.

OBJECTIVE: To examine the long-term maintenance of a previously reported behavioral counseling intervention to reduce asthmatic children's exposure to environmental tobacco smoke (ETS). PARTICIPANTS: Families of asthmatic children (6 to 17 years), including at least 1 parent who smoked in the home, recruited from 4 pediatric allergy clinics. DESIGN: Participants were randomized to 1 of 3 groups: behavioral counseling to reduce ETS exposure, self-monitoring control, and usual medical care control. Counseling concluded at month 6, and the original trial ended at month 12. Two follow-up interviews occurred at months 20 and 30. MEASUREMENTS & RESULTS: The originally reported analysis of baseline to 12 months was reanalyzed with a more robust restricted maximum likelihood procedure. The 2-year follow-up period was analyzed similarly. Significantly greater change occurred in the counseling group than the control groups and was sustained throughout the 2 years of follow-up. Further exploratory analyses suggested that printed counseling materials given to all participants at month 12 (conclusion of the original study) were

(Abstracts continued on Page 319)

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Original Contributions

Volume Support Ventilation in Infants and Children: Analysis of a Case Series

Heather T Keenan MD and Lynn D Martin MD

BACKGROUND: Volume support ventilation (VSV) augments a patient's spontaneous respiratory efforts by delivering a patient-triggered, pressure-limited, flow-cycled breath, monitors breath-to-breath changes in the pressure-volume relationship of the respiratory system during spontaneous ventilation, and regulates airway pressure to maintain a predetermined minute ventilation. Use of VSV has not been previously reported; consequently, we report our experience with VSV in infants, children, and adolescents. **PATIENTS & METHODS:** All patients requiring mechanical ventilation in the Pediatric and Infant Intensive Care Units from 12/94 through 12/95 were retrospectively reviewed, and those receiving VSV identified. Demographic data and ventilatory parameters were obtained from the medical record immediately before starting, upon initiation of, and at the conclusion of VSV. **RESULTS:** Twenty children were mechanically ventilated with VSV for part or all of their ICU course. Peak inspiratory pressure (PIP) and set tidal volume (V_T) decreased with the change to VSV [mean (SD): PIP 28.5 (6.3) vs 22.9 (7.3) cm H_2O, $p < 0.05$; V_T 9.6 (2.7) vs 7.8 (1.9) mL/kg, $p < 0.05$]. PIP, mean airway pressure ($\overline{P}_{aw}$), and V_T decreased between initial and final VSV settings [PIP 22.9 (7.3) vs 18.1 (7.1) cm H_2O, $p < 0.05$; $\overline{P}_{aw}$ 9.2 (6.3) vs 7.9 (3.0) cm H_2O, $p < 0.05$; V_T 7.8 (1.9) vs 6.0 (2.1) mL/kg, $p < 0.05$]. Eighteen patients (90%) were successfully weaned to extubation. Only 12 of 20 patients (60%) were successfully extubated from VSV. Reasons for failure to wean or extubate from VSV included clinician unfamiliarity (5 patients), paralyzed hemidiaphragm, airway obstruction/tracheal stenosis, and altered respiratory drive (1 each). Four of 5 patients (80%) not extubated from VSV due to clinician unfamiliarity were successfully extubated within a few hours of changing to a different mode of ventilation. **CONCLUSION:** Although experience with VSV is limited and nuances must be mastered, theoretic advantages (lower airway pressures, enhanced patient comfort, appropriate respiratory muscle use, and ease of clinical assessment) may make it useful in selected patients. Further study appears warranted. [Respir Care 1997;42(3):281-287]

Introduction

Over the past 20 years, many new modes of ventilation have been made available with theoretic advantages for ventilating or weaning the patient from mechanical ventilation. In the 1970s, intermittent mandatory ventilation (IMV) was introduced with the Babybird ventilator as a means to ventilate infants[1] and was first popularized by Downs[2] as a method of weaning from mechanical ventilation in adult patients. IMV,

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Dr Keenan is Fellow, Pediatric Critical Care Medicine, Department of Anesthesiology, and Dr Martin is Assistant Professor, Departments of Anesthesiology and Pediatrics, University of Washington School of Medicine. They are also associated with the Department of Anesthesia and Critical Care, Children's Hospital and Medical Center—Seattle, Washington.

Reprints & Correspondence: Lynn D Martin MD, Department of Anesthesia & Critical Care, Children's Hospital & Medical Center, PO Box 5371, Seattle WA 98105.

as classically presented, is a machine triggered, flow-limited, time-cycled mode of ventilation that provides continuous airflow to allow spontaneous breaths.[3] Therefore, it provides a guaranteed level of minute ventilation while permitting additional spontaneous breaths with minimal imposed work of breathing. The development of microprocessor technology enabled subsequent generations of ventilators to sense a patient's inspiratory effort and to deliver synchronized mechanical breaths (SIMV). Theoretic advantages of SIMV were improved patient synchrony with the ventilator and maintenance of respiratory muscle function and coordination.[3] It was hoped that this method of ventilation would allow a smooth transition from controlled mechanical ventilation to spontaneous ventilation. However, the inspiratory and expiratory valves of the synchronized microprocessor ventilators imposed significant increases on the patient's work of breathing.[4-6]

Pressure support ventilation (PSV) was added to many ventilators in the 1980s. PSV is a patient-triggered, pressure-limited, flow-cycled method of ventilation designed to overcome the resistance of the endotracheal tube and respiratory circuit. Thus, it decreases the patient's work of breathing by pressure assisting each breath.[7] Some investigators have suggested that the low-pressure-, high-volume-change work per breath provided by PSV enhances endurance conditioning of the diaphragm as opposed to the high-pressure, low-volume strength conditioning of the diaphragm seen with all other modes of assisted ventilation.[8] As the patient recovers, the support could gradually be decreased to exercise the respiratory muscles. This endurance conditioning theoretically may prove beneficial to the diaphragm and aid in the weaning of patients from the ventilator.[9] Furthermore, this mode of ventilation was thought to be more comfortable because the machine allows patients to set their own respiratory rate, inspiratory time, and flow.

Volume support ventilation (VSV), introduced in the 1990s with the Siemens Servo 300 mechanical ventilator (Siemens-Elema AB, Solna, Sweden), is an option for mechanically ventilating patients designed to augment a patient's spontaneous respiratory efforts by delivering a patient-triggered, pressure-limited, flow-cycled breath. Like pressure support, the patient determines not only the respiratory frequency but also the inspiratory time and flow. However, VSV has the capability of monitoring breath-to-breath changes in the pressure-volume relationship of the respiratory system and regulating the delivered airway pressure to achieve a minimum minute ventilation ($\dot{V}_E$). To our knowledge, the use of VSV has not been previously reported in adults or children; hence, the aim of this descriptive, retrospective case series is to present our experience with VSV in infants, children, and adolescents recovering from cardiorespiratory failure.

Patients & Methods

Patients who received VSV in the Pediatric Intensive Care Unit (PICU) and the Infant Intensive Care Unit (IICU) at Children's Hospital and Medical Center were retrospectively identified by review of all respiratory flow sheets from December 1994 to December 1995. Information extracted from the medical record of identified patients included age, weight, diagnosis, and endotracheal tube size. Information regarding the patient's ventilatory course was also obtained, including total days ventilated, days on VSV, problems with VSV, and outcome of attempted extubation from VSV. If the patient was not successfully extubated from VSV, the reason was noted. Successful extubation was defined as a 24-hour period without mechanical ventilation following extubation. Mean airway pressure ($\overline{P}_{aw}$), peak inspiratory pressure (PIP), and positive end-expiratory pressure (PEEP), $\dot{V}_E$, tidal volume (V_T), respiratory rate, and arterial blood gas tensions (when available) were recorded immediately prior to changing to VSV, on the initial VSV settings, and immediately prior to extubation. Measurements of airway pressures, inspiratory V_T, and $\dot{V}_E$ were taken from the Servo 300 digital display. Data from all ventilators were corrected for tubing compliance, temperature, and pressure; and, consistent with institutional policy, the ventilators received a pre-use function test prior to each use. In addition, all Siemens Servo 300 ventilators underwent a formal calibration every 1,000 hours as specified by the manufacturer. In all cases, the decisions to use VSV as well as the initial transition to and subsequent weaning with VSV were left to the discretion of the bedside care team. In general, our practice is to limit the use of volume support to infants and children with intact respiratory drive and those with normal lung mechanics (ie, those intubated for airway protection) or those in the recovery phase of their respiratory illness.

The hypothesis that there was no difference between the measured variables when the patient was changed to volume support or between the initial and final volume support settings was tested using the Wilcoxon matched pair signed rank test. A non-parametric test was chosen for non-normally distributed data. Significance was defined as $p < 0.05$. The Bonferroni method was used to adjust for multiple comparisons.[10]

Results

Over the study period, 591 patients were ventilated for 4,431 total ventilator days in the two ICUs. Of the 591 patients, 20 received VSV for a total of 119 days. Their clinical characteristics are presented in Table 1. The wide range of ages, weights, and diagnoses are representative of the population in the PICU and IICU. Several different modes of ventilation were used in patients prior to the change to VSV. A composite of data is displayed in Table 2.

Ventilation variables are displayed in Table 3. PIP and set V_T decreased after patients were changed to VSV from the previous mode. The PIP, $\overline{P}_{aw}$, and V_T between the initial VSV value and the VSV value prior to extubation also decreased. The respiratory rate increased between the initial and final

Table 1. Clinical Characteristics of Patients Receiving Volume Support Ventilation (VSV).

Patient Number	Age	Weight (kg)	Diagnosis/Condition	Mode of Ventilation Prior to VSV	Duration of Ventilation (days) VSV	Duration of Ventilation (days) Total
1	9 mo	5.7	Postoperative bi-directional Glenn procedure for hypoplastic left heart	SIMV-PL*	5	7
2	12 yr	36.0	Ulcerative colitis, enterocutaneous fistula, sepsis syndrome	SIMV-PL	2	9
3	11 yr	54.5	Near-drowning, acute respiratory distress syndrome	SIMV-PL	2	9
4	6 mo	5.6	Postoperative bi-directional Glenn procedure for hypoplastic left heart	SIMV-VL	6	12
5	6 wk	2.2	34-wk premature, RSV bronchiolitis, bronchopleural fistula	HFOV, PRVC	3	13
6	6 yr	24.2	Near-drowning, pulmonary edema	PRVC	1	1
7	5 yr	32.2	Necrotizing fasciitis, acute respiratory distress syndrome	HFOV, PRVC	2	13
8	13 yr	75.0	Staphylococcal sepsis, toxic shock syndrome, necrotizing fasciitis	SIMV-VL	11	16
9	11 yr	30.0	Spastic quadriplegia, aspiration pneumonia	SIMV-VL	7	8
10	5 mo	5.7	CHARGE association, micrognathia, tracheal stenosis	SIMV-PL	1	9
11	8 mo	6.6	Postoperative bi-directional Glenn procedure, scimitar syndrome, single ventricle	SIMV-PL	4	>60
12	10 d	3.6	Postoperative repair of tracheoesophageal fistula	SIMV-PL	1	22
13	3 yr	12.0	Post-Fontan procedure for double-outlet RV & cardiac arrest	SIMV-VL	1	19
14	19 mo	12.6	Ebstein's anomaly, influenza A, staphylococcal endocarditis	HFOV, SIMV-PL	3	21
15	19 yr	100.0	Repair of ascending aortic aneurysm, Marfan's syndrome	SIMV-VL	3	6
16	13 yr	39.4	Meningococcemia, septic shock	PRVC	1	4
17	2 mo	0.9	26-wk AGA premature, necrotizing enterocolitis	SIMC-PL	1	50
18	5 mo	8.0	Infantile botulism	—	53	53
19	11 yr	77.0	Postoperative repair of C-6 fracture quadriplegia from motor vehicle accident	SIMV-PL	3	21
20	27 mo	11.3	Post-Fontan procedure for tricuspid atresia	SIMV-PL	4	7

*SIMV-PL = synchronized intermittent mandatory ventilation, pressure-limited; SIMV-VL = synchronized intermittent mandatory ventilation, volume-limited; RSV = respiratory syncytial virus, HFOV = high-frequency oscillatory ventilation; PRVC = pressure-regulated volume control; CHARGE = syndrome including developmental defects of the eye Coloboma, **H**eart disease, choanal **A**tresia, **R**etarded growth and developmental and/or CNS anomalies, **G**enital hypoplasia, and **E**ar anomalies and/or deafness; RV = right ventricle; AGA = appropriate for gestational age.

Table 2. Descriptive Data on Patients Who Received Volume Support Ventilation (VSV)

Characteristic	Median	Range
Age, months	31	10 - 221
Weight, kilograms	12.0	0.9 - 100.0
Total ventilator days	12	2 - 53
Total days of VSV	2.5	0.5 - 53.0

Table 3. Changes in the Measured Ventilator Parameters During Volume Support Ventilation (VSV)

Parameter	Prior to VSV	Initial VSV	Final VSV
Peak airway pressure (cm H_2O)	28.5 (6.3)*	22.9 (7.3)†	18.1 (7.1)‡
Mean airway pressure (cm H_2O)	12.1 (5.9)	9.2 (6.3)	7.9 (3.0)‡
Total minute ventilation (mL/kg/min)	350 (279)	304 (239)	359 (368)
Inspiratory tidal volume (mL/kg)	9.6 (2.7)	7.8 (1.9)†	6.0 (2.1)‡
Total respiratory rate (f)	23.5 (15.0)	26.6 (13.2)	35.0 (13.7)‡

* Values are mean (SD).

$^{\dagger}p < 0.05$ between prior to VSV and initial VSV values.

$^{\ddagger}p < 0.05$ between initial VSV and final VSV values.

VSV setting. The increased respiratory rate coupled with the decreased V_T resulted in equivalent $\dot{V}_E$ during initial VSV and prior to extubation. Paired arterial blood gas values were not available for all patients, but for the 11 patients for whom they were available, no significant difference was seen in pH [pre-VSV 7.39 (0.07) vs VSV 7.43 (0.06) $p = 0.06$], P_{aCO_2} [pre-VSV 46 (10) vs VSV 44 (7) torr, $p = 0.47$], and P_{aO_2} [pre-VSV 87 (26) vs VSV 81 (28) torr, $p = 0.49$] from the previous mode of ventilation to VSV.

Eighteen of the patients (90%) were successfully weaned to extubation and remained extubated. Of those, 12 were extubated from VSV. Reasons for failure to be weaned or extubated from VSV are shown in Table 4. Four patients weaned well on VSV but were changed to an alternate mode 5-24 hours prior to extubation because the clinician was unfamiliar with judging readiness for extubation during VSV. All of these patients were successfully extubated. One patient required reintubation shortly after elective extubation from VSV. In retrospect, this patient had increasing PIP, $\bar{P}_{aw}$, and respiratory rate prior to extubation. Therefore, he did not meet the usual criteria for extubation and not surprisingly required immediate reintubation. One patient with a paralyzed left hemidiaphragm secondary to iatrogenic phrenic nerve injury did well on VSV, but eventually required a tracheotomy and home ventilation. One patient extubated from VSV developed imme-

Table 4. Description of Patients for whom Volume Support Ventilation (VSV) Was Considered a Failure

Patient Number	Age	Diagnosis/Condition	Type of Failure*	Etiology of Failure	Treatment
9	11 yr	Spastic quadriplegia, aspiration pneumonia	Extubation	Clinician preference	Switched to SIMV+PSV[†] 24 hours prior to extubation
10	5 mo	CHARGE association, micrognathia, tracheal stenosis	Extubation	Airway obstruction	Tracheotomy
11	8 mo	Postoperative bi-directional Glenn procedure, scimitar syndrome, single ventricle	Extubation	Paralyzed left hemi-diaphragm	Tracheotomy and chronic home ventilation
12	10 d	Postoperative repair of tracheo-esophageal fistula	Extubation	Respiratory distress	Switched to SIMV following immediate re-intubation
13	3 yr	Post-Fontan procedure for double-outlet RV and cardiac arrest	Weaning	Neurologic injury with inconsistent ventilatory drive	Switched to SIMV until resolution of neurologic injury and extubation
17	2 mo	26-wk AGA premature, necrotizing enterocolitis	Extubation	Clinician preference	Switched to PSV 6 hours prior to extubation
19	11 yr	Postoperative repair of C-6 fracture quadriplegia from motor vehicle accident	Extubation	Clinician preference	Switched to PSV 5 hours prior to extubation
20	27 mo	Post-Fontan procedure for tricuspid atresia	Extubation	Clinician preference	Switched to SIMV+PSV, 16 hours prior to extubation

*Weaning failure is defined as an intolerance of VSV, extubation failure defined as not extubated from VSV.

SIMV+PSV = synchronized intermittent mandatory ventilation plus pressure support ventilation, CHARGE = syndrome including developmental defects of the eye **C**oloboma, **H**eart disease, choanal **A**tresia, **R**etarded growth and developmental and/or CNS anomalies, **G**enital hypoplasia, and **E**ar anomalies and/or deafness, RV = right ventricle.

diate airway obstruction requiring reintubation. Subsequent extubation attempts from other modes of ventilation for this patient also proved fruitless, and he ultimately required a tracheotomy. Only one patient did not tolerate VSV. This patient had an inconsistent respiratory drive secondary to an evolving neurologic injury. His fluctuating drive resulted in repeated activation of the backup PRVC mode and associated alarm because of apnea. This patient was switched to SIMV until stabilization of the neurologic injury and ventilatory drive, at which time extubation occurred without problems.

Discussion

This is the first published report of the use of VSV for mechanical ventilation in either children or adults. This new mode of ventilation, offered on the Siemens Servo 300 ventilator, is designed to support spontaneously breathing patients on mechanical ventilation. It has been described by some as adaptive pressure support.[11] In this mode of ventilation, V_T is used as a feedback control for continuously adjusting the pressure support level. The ventilator initiates VSV by delivering a series of 4 test breaths of incrementally greater pressure synchronized to the patient's inspiratory effort. The delivered V_T is measured and respiratory system compliance calculated. The calculated system compliance is used to manipulate the pressure support level until the target V_T is delivered. The maximum breath-to-breath pressure change once the target V_T is achieved is limited to 3 cm H_2O and can range from 0 cm H_2O above the set PEEP to 5 cm H_2O below the high-pressure alarm limit. Breaths are primarily terminated when inspiratory flow decreases to 5% of the initial peak flow and secondarily when inspiratory time reaches 80% of the set total cycle time. The control logic of this mode of ventilation is depicted in Figure 1.[12]

Patients who theoretically may benefit from VSV include those who are awake and ready to wean; those who have an intact ventilatory drive but need airway protection; those who spontaneously breathe but require PEEP; and those being weaned from prolonged ventilatory support. In this mode of ventilation, the patient determines the inspiratory time, flow, and frequency, while the clinician sets a minimum $\dot{V}_E$ goal for the patient to meet. This $\dot{V}_E$ goal is approximated by initially using 75-90% of the V_T and respiratory rate that the patient is receiving in his current mode of ventilation and then titrating the goal to patient comfort. The ventilator attempts to deliver the set $\dot{V}_E$ goal at the lowest possible pressure. Indeed, in this series of patients, decreases of PIP and set V_T were seen when the ventilator mode was changed to VSV while the same delivered $\dot{V}_E$ was maintained.

Two patients developed upper lobe atelectasis following the change to VSV. On bronchoscopic examination, one of these patients proved to have compression of the left mainstem bronchus by vascular structures. The other required increased PEEP to reinflate the lobe. This experience suggests that the clinician needs to be aware that the amount of support given to a spontaneously breathing patient with VSV may

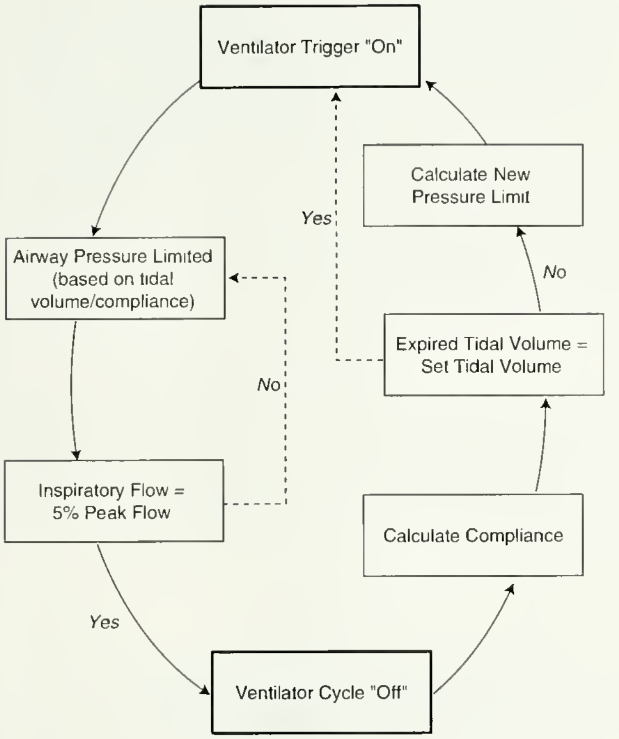
Ventilator Trigger "On"
Calculate New Pressure Limit
Yes
No
Airway Pressure Limited (based on tidal volume/compliance)
Expired Tidal Volume = Set Tidal Volume
No
Inspiratory Flow = 5% Peak Flow
Calculate Compliance
Yes
Ventilator Cycle "Off"

Fig. 1. The control logic of volume support ventilation on the Siemens 300 ventilator (modified from Reference 11).

be less than that afforded by other modes. This may result in a larger drop in $\overline{P}_{aw}$ than is desirable, but this can be compensated for by slightly increasing the PEEP in order to sustain the $\overline{P}_{aw}$ and maintain sufficient lung expansion.

Weaning in this mode of ventilation can occur actively or passively. With active weaning, the clinician gradually decreases the set V_T or $\dot{V}_E$ goal. When a physiologic spontaneous V_T (ie, 3-6 mL/kg) has been achieved, the patient is extubated. If the patient becomes tachypneic or distressed, the decrease in ventilatory assistance has been too great and the volume goal should be increased. With passive weaning, the ventilator 'self-weans' the patient as the patient's muscle strength and respiratory mechanics improve (ie, as the patient generates a larger percentage of each V_T and, consequently, the inspiratory pressure generated by the ventilator decreases. The clinician sees a gradual decrease in PIP and $\overline{P}_{aw}$. We have found that the patient typically can be safely extubated when his set V_T is 3-6 mL/kg, PIP $\leq$ 20 cm H_2O, respiration appears comfortable, and oxygenation is adequate. In our series, PIP, $\overline{P}_{aw}$, and set V_T fell as the patient moved toward extubation. All patients who met these goals tolerated extubation well.

One potential advantage of VSV is patient comfort. A commonly encountered problem in the small pediatric patient is failure to achieve synchronization between the ventilator and the infant. In fact, several patients in this series developed acute respiratory distress as SIMV rates were decreased; thereby, halting any further weaning attempts. These difficult-to-wean patients were then switched to VSV and progressed to extubation without difficulty. In the past with other modes of ventilation, sedatives and occasionally muscle relaxants have been used to overcome asynchrony. However, when the patient is ready to be weaned from mechanical ventilation, we have found it sometimes difficult to achieve the right balance between optimal sedation and adequate ventilatory drive. By allowing the infant or child to set his or her own respiratory rate, inspiratory time, and flow (ie, breathing pattern), this mode theoretically should reduce asynchrony between patient and ventilator. In addition, the microprocessor circuitry of this ventilator (Siemens Servo 300) provides a rapid response to a small change in inspiratory flow (ie, flow-triggering). In one trial of the Servo 300,[13] the majority of patients were able to trigger the ventilator in the first 40% of contraction of the diaphragm as measured by electromyogram. Therefore, it is responsive to infants and small children with rapid respiratory rates and should decrease the work of breathing.[14] These factors may decrease the child's need for sedation and aid in weaning.

Although only a minority of pediatric patients are difficult to wean from mechanical ventilation, some children with respiratory failure are intubated and ventilated for extended periods of time and may require muscle relaxants. These extended periods of decreased muscle use can lead to atrophy and weakness that may result in an even more prolonged weaning period. There is currently no consensus on whether complete muscle rest interspersed with periods of strenuous activity or continuous low levels of respiratory work are more advantageous for training respiratory muscle.[15] However, a rapid respiratory rate has been shown to be a sensitive indicator of muscle fatigue,[16,17] and can be used as an indicator of excessive work of breathing. VSV may be advantageous in this situation because it allows respiratory muscle work to be varied depending on the clinical status of the patient. In addition, several studies suggest that the change in pressure-volume load characteristics that occurs during PSV enhances endurance conditioning of the diaphragm.[8,18] Presumably, these same benefits would be seen with VSV, which has similar pressure-load characteristics.

There are several potential disadvantages to VSV. Our experience suggests that the clinician's 'learning curve' for this new mode of ventilation can be steep. Difficulties have been encountered both in deciding on the initial ventilator settings and in clinician comfort with judging readiness for extubation. The former problems have included setting the $\dot{V}_E$ goal too high or too low. A high $\dot{V}_E$ and V_T goal drives down the patient's arterial carbon dioxide tension and can cause long respiratory pauses. An initial $\dot{V}_E$ and V_T goal that is too low can cause the patient to become distressed and tachypneic. These problems, if recognized, are easily corrected by changing the $\dot{V}_E$ and V_T goal while monitoring the patient's breathing pattern. Another common problem occurs when setting the respiratory rate. In VSV, the set respiratory rate serves three functions: (1) with the V_T it is used by the ventilator to determine the minimum $\dot{V}_E$; (2) it sets the respiratory cycle time and provides a secondary mechanism for inspiratory termination; and (3) it serves as the ventilator rate should the patient become apneic and the backup mode (PRVC) becomes activated. If the minimum respiratory rate is set too high, the $\dot{V}_E$ goal is also probably too high. If the patient slows his respiratory rate below the minimum rate, the support provided by the ventilator increases to meet the $\dot{V}_E$ goal. This results in larger V_Ts interspersed with long pauses. The pauses can exceed the apneic limit of the ventilator and result in default activation of the backup PRVC mode. Therefore, the respiratory rate needs to be set below the lowest rate that the patient is expected to breathe during quiet or resting states.

Another difficulty has been clinician discomfort with judging the patient's readiness for extubation. Although criteria that indicate readiness for extubation in infants have been established (crying vital capacity of >15 mL/kg and a maximum inspiratory pressure of ≥ -45 cm H_2O),[19] many clinicians continue to rely on their subjective bedside assessment of patient comfort. This mode lends itself well to this type of assessment.

Patients must have a consistent respiratory drive to tolerate VSV. Patients who are heavily sedated or those with an impaired neurologic status are not good candidates for VSV. Application in neonates with periodic breathing of the newborn can also be problematic. The neonatal setting on the Servo 300 allows only a 10-second apnea before changing to the backup mode (PRVC). The pediatric setting increases the apnea period to 15 seconds, potentially overcoming this problem.

Based on our experience with this series, we suggest that the clinician aim for 75-90% of the patient's current $\dot{V}_E$, increase PEEP as needed to keep the $\overline{P}_{aw}$ from falling too precipitously, and set the backup rate slightly below the slowest rate at which one would expect the patient to breathe during quiet, resting states. These initial settings may then be adjusted to achieve the respiratory rate and pattern most comfortable for the patient.

The limitations of the retrospective analysis of this case series must be recognized. Care was not delivered by previously established protocol, the total number of patients was small, data were incomplete, no concurrent control patients were available for comparison, and the selection criteria for the use of VSV was solely by provider preference, which may lead to a biased patient population not representative of typical PICU and HCU populations. Despite these limitations, the analysis and reporting of our clinical experience may provide valuable information that can be used to assist further investigations.

Conclusion

Despite the initial challenge of understanding a new mode of ventilation and its appropriate clinical application, VSV has theoretic advantages that may make it a useful mode in selected patients. Potential advantages include a fixed minimum minute ventilation, lower airway pressures, enhanced patient comfort, appropriate respiratory muscle use, and ease of clinical assessment. Although this report shares the limitations of descriptive, retrospective studies, it illustrates that VSV can be successfully used in a subset of the PICU-HCU population. Although clinical experience with VSV is limited, its theoretic advantages suggest that further study of its clinical application is warranted.

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Comparison of Two Methods for Securing the Endotracheal Tube in Neonates

Teresa A Volsko RRT and Robert L Chatburn RRT

INTRODUCTION: Securing the uncuffed endotracheal tubes of infants requiring mechanical ventilation is imperative to avoid the complications that often accompany unplanned extubation. We evaluated the effect of two methods of securing the endotracheal tube on the incidence of spontaneous extubation. MATERIALS & METHOD: We prospectively studied the incidence of accidental extubation in 244 infants, meeting predetermined criteria, who were admitted to our Level-2 neonatal intensive care unit. Oral endotracheal tubes were secured by conventional taping or by an oral fixation device (the Logan Bow, ie, nuchal arch). The device was applied depending on availability. RESULTS: Fewer patients were accidentally extubated with the Logan Bow (21.3% vs 58.7%, $p < 0.0001$) and there were fewer extubations/100 ventilator days (2.1 vs 3.0). When stratified by weight, the difference in extubation weights was significant only for patients weighing ≤ 1.5 kg. CONCLUSION: The use of the Logan Bow to secure uncuffed endotracheal tubes may reduce the unplanned extubation rate of mechanically ventilated low birthweight infants. [Respir Care 1997;42(3):288-291]

Introduction

Successful management of mechanically ventilated infants depends on the caregivers' ability to secure and maintain an artificial airway. Accidental or spontaneous extubations interrupt airway patency and may complicate the patient's course. Adverse patient outcomes, such as laryngospasm, edema of laryngeal structures, aspiration of pooled secretion and/or gastric contents, acute hypoxia, and hypercarbia often can result.[1] Complications arising from repeated spontaneous extubations and subsequent reintubations may include profound hypoxia, acidosis, cardiopulmonary compromise, subglottic stenosis, laryngotracheal ulceration, necrosis, and granuloma.[2] The sequelae of intraventricular hemorrhage and pulmonary hypertension may ensue in conjunction with an hypoxic or acidotic state.[3]

Factors associated with spontaneous extubation are varied. Numerous conditions accompanying unplanned extubations including airway anatomic structure, time of day, bedside care, patient position, use of limb restraints, sedation, infant's size and activity level, amount of oral secretions, and type and integrity of the securing device.[4]

A common denominator, however, is the importance of securing the uncuffed endotracheal tube in order to minimize these negative sequelae. The purpose of this study was to evaluate the effect of two endotracheal tube taping methods on the incidence of spontaneous extubations. We hypothesized that the use of the Logan Bow (small size: Rusch Model 395429, Rusch Co, Duluth GA, and medium size: Storz Model N5958, Storz Instruments, Manchester MO) would reduce the

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Ms Volsko is Clinical Specialist, Respiratory Care Department, MetroHealth Medical Center, Cleveland, Ohio. She was the Coordinator NICU/Pediatrics, Respiratory Services, St Elizabeth's Health Center, Youngstown, Ohio when the research was performed. Mr Chatburn is Director, Respiratory Care Department, University Hospitals of Cleveland, and Instructor, Department of Pediatrics, Case Western Reserve University—Cleveland, Ohio.

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Reprints & Correspondence: Teresa Volsko RRT, Respiratory Care Department, MetroHealth Medical Center, 2500 MetroHealth Dr, Cleveland OH 44109-1998.

incidence of spontaneous extubations in orally intubated infants compared to a conventional taping method.

Materials & Method

We studied a convenience sampling of 244 infants admitted to our Level-II special care nursery who required intubation and mechanical ventilation. Excluded from the study were (1) infants who required limb restraints, sedation, or paralytic drugs that inhibit or restrict activity; (2) those with suspected or confirmed neurologic impairment that prevented purposeful movements; (3) those scheduled for transfer to tertiary care facilities; and/or (4) those requiring nasotracheal intubation or mechanical ventilatory support for < 72 hours or > 30 days.

Infants enrolled in this prospective study had their oral endotracheal tube affixed by one of two methods. The method used to secure the endotracheal tube depended on the availability of the device.

Logan Bow

The Logan Bow (or nuchal arch), an oral fixation device, was described by Budd[5] in 1982 and by Franck[6] in 1992 as an endotracheal tube securing method (Fig. 1) For our study, infants orally intubated and requiring positive pressure ventilation had their oral endotracheal tubes affixed by a Logan Bow appropriate for their size when the device was available. Small-sized Logan Bows were used for infants who weighed ≤ 700 g and medium for those weighing > 700 g.

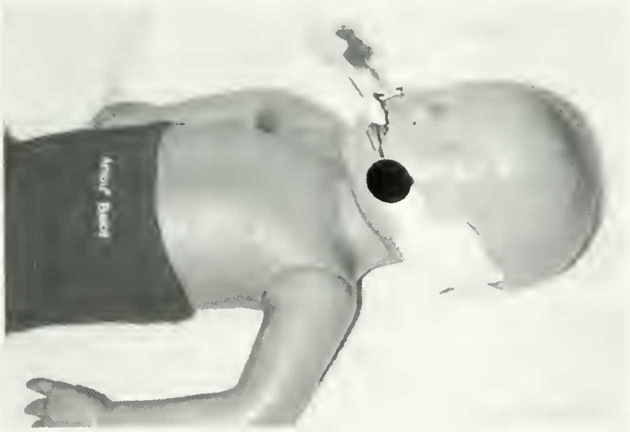

Fig. 1. The Logan Bow (or nuchal arch) as an endotracheal tube securing method.

Preparation of the Logan Bow was necessary prior to application of the device. A head strap was devised by placing a piece of adhesive tape through the center of each ring, located at either end of the Logan Bow and securing the tape to itself. The adhesive tape strips were of uniform width, 1 in. The length to which they were cut averaged 4 in., varying according to cranial diameter at the occiput.

Circular temperature probe covers (Critter Covers, Airshields/Vickers Medical, Hatfield PA) were affixed to either side of the rings at the ends of the Logan Bow to protect the infant's face from indentation, irritation, or injury from contact with the metal ends of the bow. Gauze pads (2" × 2") were used to cover the infant's ears prior to fitting the device over the face, allowing the covered ends to rest upon the cheeks. Placement was inspected to ensure that the ears were shielded from adhesive tape and that the device was not squeezing the face or bending or kinking the endotracheal tube. The adhesive tape strips were wrapped around the occiput. Care was taken to guard the infant's neck from the tape. A slip knot, with umbilical ties, secured the endotracheal tube to the bow. The patency of the oral endotracheal tube was verified by unobstructed passage of a suction catheter through the tube. Two pieces of adhesive tape were used to hold the slip knot in place. Each piece, (1/4" wide and 3" long) was wrapped around the bow and the endotracheal tube anchoring them together.

Conventional Taping Method

If a bow of the proper size was not available, the endotracheal tube was secured to the infant's face by conventional taping. Prior to the application, 2 strips of adhesive tape, (approximately 1/2" wide and 4" long) were cut. A horizontal slit, three fourths of the length of the strip was made, yielding a Y-shaped piece. The infant's skin was prepared by thoroughly drying the perinasal and cheek area. The clean, dry areas were then painted with tincture of benzoin. The opening of the Y-shaped adhesive strip was centered around the oral endotracheal tube. The top portion of the strip was affixed to the infant's upper lip. The bottom or lower portion of the tape was applied to the endotracheal tube in a counterclockwise fashion. The second adhesive strip was aligned against the tube and adhered to the perinasal area similarly. Using a clockwise rotation, the lower portion of the tape strip was wrapped around the infant's tube (Fig. 2). The patency of the lumen was verified by unobstructed passage of a suction catheter, through the endotracheal tube.

The tape, associated with either method, was changed when loss of adhesive allowed slippage of the tube. Retaping was done at the discretion of the respiratory therapist or nurse.

Statistical Analysis

Patients were grouped by weight range. The percent extubated/group was calculated as the number of patients who had at least one accidental extubation divided by the number of patients in the group. This allowed comparison of extubation data using the Fisher Exact test. One-tailed p values were reported and considered significant at the 0.05 level (2-tailed values are usually estimated by doubling the 1-tailed values).[7]

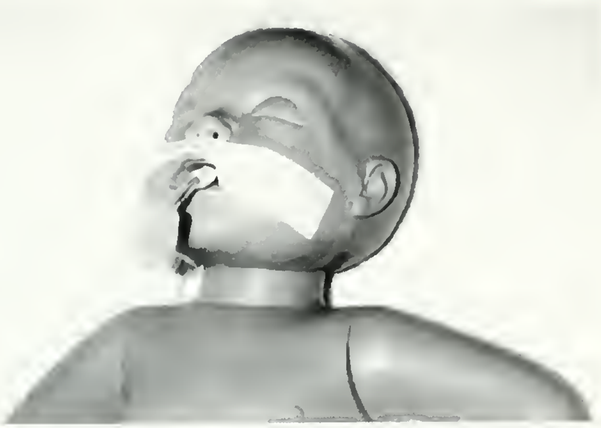

Fig. 2. Adhesive tape as an endotracheal tube securing method.

A spontaneous extubation was defined as an unplanned dislodgment of the oral endotracheal tube without regard to the precipitating event (eg, extubation during retaping, repositioning, routine bedside care, or in conjunction with a therapeutic procedure), but not associated with events that might contribute to clinical deterioration, such as bronchospasm or mucus plugging.

Calculation of percent extubated as described earlier ignores data for patients who had multiple extubations. To include all data, we calculated the total number of extubations/100 ventilator days. However, this index could not be compared statistically.

Results

Data obtained during the 18-month study period are shown in Table 1. There was no statistically significant difference in accidental extubation rates for infants weighing > 1.5 kg. For infant's weighing 0.5-1.5 kg, the group with the conventional taping had an average accidental extubation rate six times that of the Logan Bow group.

Table 1. Comparison of Outcome of Tube Stabilization Methods, Stratified by Infant Weight

	Logan Bow Total Patients = 89		Adhesive Tape Total Patients = 155		
Weight Range (kg)	No. in Category	Extubated n (%)	No. in Category	Extubated n (%)	p value
0.50-1.00	12	2 (16.7)	55	45 (81.8)	< 0.0001
1.01-1.50	19	2 (10.5)	31	25 (80.6)	< 0.0001
1.51-2.00	19	6 (31.6)	15	4.(26.7)	0.53
2.01-2.50	19	2 (10.5)	21	3 (14.3)	0.55
2.51-4.40	20	7 (35.0)	33	14 (42.4)	0.40
Total Patients	89	19 (21.3)*	155	91 (58.7)	< 0.0001

*Extubations/100 ventilator days with Logan Bow = 2.1, with adhesive tape = 3.0.

Discussion

Spontaneous extubations have been associated with profound complications in infants and children, and devastating consequences have resulted from repeated intubations after spontaneous extubations.[8]

Although various factors contribute to spontaneous extubations among infants, the shortness of the infant's trachea coupled with the narrow range for optimal endotracheal tube placement particularly facilitate accidental extubations.[9] The tip of the uncuffed endotracheal tube should lie 2 cm below the vocal cords and 1-2 cm above the carina, which leaves little room for movement without the risk of malposition.[10] It has been reported that extension of the head by a term infant can withdraw the tube by 3.5 cm and flexion can advance it by 1.8 cm.[11] Therefore, it is imperative that the tube movement associated with head movement and positioning be minimized. A number of fixation methods have been described in the literature.[4,12,13] The method chosen should not interfere with access to the tube and should be easy to apply and remove.[12] Durability of the device is of equal importance. It should ensure the stability of the endotracheal tube in the face of inadvertent traction from head movement, ventilator tubing, or patient activity.[13] Constant exposure to oral secretions reduces the adhesiveness of tape around the tube and can contribute to accidental extubation.

The Logan Bow is among a number of devices reported to circumvent this problem.[4] In our study, an adhesive head sling anchored the device and the oral endotracheal tube. Stability of the artificial airway was provided in the face of traction imposed by the ventilator circuitry and patient movement. In addition, access to the oropharyngeal cavity for nursing care was unobstructed. Gauze pads and temperature probe covers shielded the infant's face from adhesive tape, minimizing skin irritation while augmenting the ease of device application and removal. The design of the Logan Bow allows the oral endotracheal tube to be secured away from the infant's mouth and face. This minimizes contact with oral secretions and reduces the risk of tube slippage and subsequent unplanned extubation.

Accidental extubation data are not reported consistently, making it difficult to compare our data with that of other studies. Kleiber and Hummel[4] have written a comprehensive review of spontaneous extubation research spanning 28 years. They found that many of the investigations lacked an operational definition for spontaneous extubation, a mechanism to identify the population studied, and a uniform reporting method. For example, one study reported the rate of spontaneous extubations as the number of extubations/100 intubated infants,[14] while another study compared the number of spontaneous extubations/100 ventilator days.[13] Some studies do not account for the acuteness of the patient's condition, the duration of intubation, or repeated accidental extubations. Our study follows the suggestions by Hummel and

Kleiber[15] in that we specifically defined spontaneous extubations. Our population was identified by weight and duration of mechanical ventilation to include infants in the acute phase of illness rather than the chronic ventilator dependent infants (defined as mechanical ventilation for > 30 days). In addition, we uniformly reported incidence statistics as the number of extubations/100 ventilator days. Intraobserver variation in measurement and reporting can result in bias and misinterpretation of results. The incidence of confusion and misinterpretation of results can be reduced when clinical comparison of uniformly reported data are presented.[16]

Conclusion

The use of a Logan Bow to secure an uncuffed endotracheal tube may reduce the incidence of unplanned extubations on infants weighing less than 1.5 kg compared to the use of tape alone.

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Bronchodilator Properties of Ketamine

Hugh S Mathewson MD and Thomas A Davis CRNA MAE

In the late 1950s, certain derivatives of cyclohexylamine were shown to have central depressant properties and to provide considerable analgesia when patients were still awake. The first of these to be introduced clinically was *phencyclidine*,[1] which produces a feeling of dissociation from the environment and indifference to painful stimuli. This came to be known as dissociative anesthesia.[2] However, emergence delirium and subsequent hallucinatory phenomena were so common that phencyclidine had to be abandoned—it later became a widely used street drug called PCP. Research continued, and after study of many similar derivatives the compound called *ketamine* was introduced and received official approval.[3]

The principal anesthetic virtue of ketamine is its benign effect on respiration and blood pressure when administered in induction doses, in contrast to the hypotensive effects often seen with thiopental and propofol, the most commonly used induction agents. For patients with unstable circulation, induction can be achieved with less risk. Also, minor procedures that can be performed with the patient awake, such as changing burn dressings, can be safely and less painfully accomplished with dissociative anesthesia. The problems of postoperative psychodysleptic effects (ie, distorted perception, hallucinations, and delusions) remain to some degree, although not nearly to the extent previously encountered with phencyclidine.[4] These untoward actions can be mitigated to some degree by premedication with benzodiazepines (diazepam, midazolam), but the risk of 'bad trips' has placed ketamine in a questionable position as a routine anesthetic induction agent.

In 1971 Bovill et al[5] reported that airway resistance is measurably decreased during ketamine induction, and bronchospasm may be abolished. Pulmonary vascular resistance is not altered, and hypoxic pulmonary vasoconstriction is not inhibited. With these observations in mind Corssen et al (1972)[6] recommended that ketamine be used in the anesthetic management of asthmatic patients. Although this has remained a landmark article, anesthesiologists were slow to adopt this regimen for fear of psychologic aftereffects.

It had long been recognized, however, that ketamine-induced psychedelic or psychodysleptic effects were less prone to occur in children than in adults. Much of the recent study of ketamine as a bronchodilator is concerned with the treatment of asthma in infants and children.[7-9] In the past decade, ketamine has received increasing recognition as an effective agent for terminating severe episodes of asthma, particularly in cases where conventional therapy has failed.[9-11] A study of particular interest in respiratory care showed that ketamine infusions tend to reduce bronchospasm in patients on mechanical ventilation.[12]

The mechanism by which ketamine decreases airway resistance is discussed in a recent review by Nehama et al.[13] One hypothesis is that ketamine increases synaptic catecholamine levels by blocking the reuptake of norepinephrine into presynaptic sympathetic neurons. It has been shown that the increase in free norepinephrine parallels the peak bronchodilatory effect of ketamine and that this effect can be diminished by β-adrenergic blockade. Ketamine also inhibits vagal outflow and may exert an anticholinergic effect on bronchial smooth muscle, although doses in the greater-than-normal range are required to demonstrate this effect.

Although clinical references have been sporadic since 1971, L'Hommedieu[14] reported 4 cases in 1987 in which intravenous ketamine was used for emergency intubation during status asthmaticus; the patients' ages ranged from 15 months to 14 years. Nehama[13] described the successful use of a ketamine infusion for an 8-month-old infant for whom the prescribed treatment of status asthmaticus had failed. These favorable reports portend a wider use of the drug; however, there are possible untoward effects unrelated to the psychological hazards. Ketamine increases upper airway reflexes and can cause increased secretions, cough, and laryngospasm. It has been recommended that the drug be given with a vagolytic agent and that a neuromuscular blocker be given if intubation is to be performed.[15] The administration of ketamine by aerosol has been suggested, but no reports of clinical studies have yet appeared.

Howton et al (1996)[16] performed a randomized, double-blind, placebo-controlled study of intravenous ketamine for

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Dr Mathewson is Professor Emeritus, Anesthesiology, and Professor, Respiratory Care Education, and Mr Davis is Assistant Professor, Nurse Anesthesia Education—University of Kansas Medical Center, Kansas City, Kansas.

treatment of acute asthma in the emergency department. An initial 0.2 mg/kg bolus of ketamine was given over a 5-minute period, followed by an infusion of 0.5 mg/kg/h. Of the first 9 patients, 6 received ketamine, and 3 became dysphoric. At this point, the bolus dose was reduced to 0.1 mg/kg for the remaining 44 cases studied. The conclusion was that, in doses low enough to avoid dysphoric reactions, the bronchodilator effect of ketamine was no greater than that obtained with standard therapy. Although there was a slight increase in patient satisfaction in the ketamine group, no clinical benefit in terms of hospital admission rate was noted.

Because the incidence of dysphoria is dose-related, studies in which patients were allowed to remain awake have tended to be conducted with lower doses of ketamine. This indicates that the therapeutic window is uncomfortably small and that titrating the drug to achieve bronchodilatation may be difficult in some cases.

No new congeners of ketamine have been advanced for clinical study in recent years. However, the ketamine molecule contains a chiral center, allowing the formation of 2 enantiomers, designated R(−) and S(+). Solutions of the drug available in the U.S.A. contain the racemic mixture. Researches have been undertaken to determine whether one isomer is superior enough therapeutically to justify its separation and marketing in pure form.[17] The S(+) isomer reportedly has about 3 times the analgesic and anesthetic potency of the R(−) derivative and is about 20% shorter acting. The relative bronchodilator actions of the isomers are yet undetermined. A recent experimental study indicates that they are equally spasmolytic against histamine-induced contraction of tracheal smooth muscle.[18]

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Graphics Corner

Jon Nilsestuen PhD RCP RRT, Robert Harwood MSA RRT, Ken Hargett BS RCP RRT, Section Editors. Submissions from readers are encouraged. Please follow the Manuscript Preparation Guide and submit to RESPIRATORY CARE Journal Office, 11030 Ables Lane, Dallas, Texas 75229.

A Patient in Status Asthmaticus: What Does the Waveform Confirm?

Robert Harwood MSA RRT, Lynda Thomas Goodfellow MBA RRT,
DiAnn Larson RRT, and Robert Aranson MD

Case Summary

A 32-year-old man with a history of severe asthma and bronchitis presented to the emergency room (ER) with shortness of breath, sinus congestion, and sputum production that had increased over the previous 2 weeks. Home medications had included albuterol by metered dose inhaler (MDI), 2 puffs 4 times daily and as needed; beclomethasone dipropionate by MDI, 2 puffs twice daily; theophylline 300 mg twice daily; and guaifenesin 2 tablets by mouth twice daily and as needed. He denied use of alcohol and tobacco.

On physical examination, the patient was found to be conscious and oriented to time, place, and person. His heart rate was regular at 136 beats/min, blood pressure 146/96 mm Hg with no pulsus paradoxus, and respiratory rate 30 breaths/min. His breathing was labored, and he was using accessory muscles. Cardiac auscultation revealed no murmurs, gallops, or rubs. Auscultation of the chest revealed bilateral inspiratory and expiratory wheezes. There was no clubbing of the fingers or toes or edema of the extremities.

In the ER, the patient was treated with albuterol by small volume nebulizer, I.V. (intravenous) aminophylline, and oxygen by nasal cannula at 4 L/min to maintain oxyhemoglobin saturation by pulse oximetry > 90%. Within 2 hours, 2 additional albuterol treatments were administered by nebulizer with no improvement. The patient was admitted and transferred to the intensive care unit. During the next 6 hours, the patient deteriorated and required an increase in the fractional concentration of delivered oxygen (F_{DO_2}) to 1.0 and the administration of methylprednisolone. Arterial blood gas analysis showed pH 7.26, P_{aCO_2} 76 torr [10.13 kPa], P_{aO_2} 197 torr [26.2 kPa], HCO_3^- 35 mEq/L, Base Excess +5 mEq/L. At this time, the patient was sedated, orally intubated with an 8.0-mm-ID endotracheal tube (ETT), and placed on a volume ventilator with the settings: synchronized intermittent mandatory ventilation (SIMV), frequency 12/min, tidal volume (V_T) 800 mL, F_{DO_2} 0.40; pressure support 10 cm H_2O; positive end-expiratory pressure (extrinsic PEEP, or $PEEP_e$) 0; peak flow 55 L/min. Following institution of mechanical ventilation, albuterol was nebulized continuously (10 mg/h). Because of restlessness, the patient was sedated with I.V. lorazepam and propofol. By the second day of mechanical ventilation, the patient required an increase in oxygen fraction to 0.80. $PEEP_e$ of 10 cm H_2O was added, but no other changes were made in ventilator settings.

Part 1

On chest radiograph, the lungs appeared hyperinflated. Tracings were obtained with a free-standing monitor (BiCore, Fig. 1.).

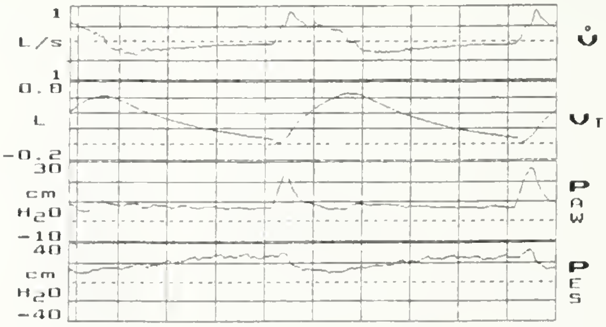

Fig. 1. Waveforms recorded during the second day of mechanical ventilation of a patient in status asthmaticus. Ventilator settings were: mode SIMV; frequency 12 breaths/min; V_T 800 mL, F_{DO_2} 0.80; pressure support 10 cm H_2O, $PEEP_e$ 10 cm H_2O.

Mr Harwood and Ms Goodfellow are faculty members of the Cardiopulmonary Care Sciences Department at Georgia State University, Ms Larson is Educational Coordinator, Respiratory Care Department, DeKalb Medical Center, and Dr Aranson is on the faculty of Emory University School of Medicine, Director of the Medical Intensive Care Unit & Respiratory Department, Grady Memorial Hospital, and Medical Director, Cardiopulmonary Care Sciences Department, Georgia State University—Atlanta, Georgia.

Reprints & Correspondence: Robert Harwood MSA RRT, Assistant Professor, Cardiopulmonary Care Sciences, Georgia State University, University Plaza, Atlanta GA 30303-3083.

How would you answer these questions?

What is the probable cause for the failure of the V_T waveform to return to baseline (dashed line)?

What has developed in this patient as seen on the flow ($\dot{V}$) waveform?

Part 2

The waveforms in Figure 1 were analyzed and the ventilator adjusted. Two additional waveforms were obtained and are shown in Figure 2.

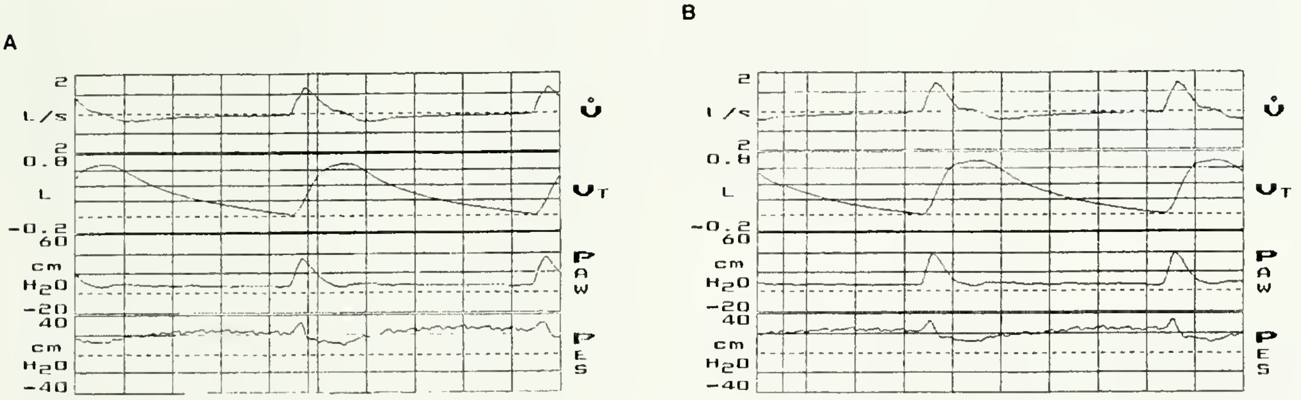

Fig. 2. A. Waveforms resulting from adjustment of the ventilator. B. Waveforms resulting from additional adjustment of the ventilator.

How would you answer these questions?

What adjustment of ventilator settings would account for the changes seen in Tracings A and B of Figure 2?

Why did the ventilator adjustment change the flow and V_T curve?

Answers and Discussion on Page 296

Answers, Part 1

V_T Waveform. Air trapping is the probable cause for the failure of the V_T waveform to return to baseline. The expiratory volume is less than the inspiratory volume. The difference between the two volumes is the trapped volume.

Auto-PEEP has developed. The end-expiratory flow does not fall to zero (ie, the flow waveform does not return to baseline) before the next positive pressure breath is delivered. Auto-PEEP (or intrinsic PEEP, PEEP$_I$) develops when the expiratory time(T_E) is too short to allow complete emptying of the lungs.

Answers, Part 2

The flow setting was increased as shown in Tracing A (from 50 L/min to 100 L/min) and further increased in Tracing B (to 120 L/min). Note in Tracing A that the increase in flow reduced the hyperinflation and PEEP$_I$. In Tracing B, the hyperinflation and PEEP$_I$ have been eliminated.

Increasing the flow reduced the inspiratory time (T_I) and increased the T_E, allowing a longer time for air to escape the lung. The reduction in trapped air is seen in the return of the flow curve to baseline before the next breath is delivered and in the return of the V_T curve to baseline. Expiratory volume now equals inspiratory volume on the V_T tracing.

Discussion

Auto-PEEP, also called occult PEEP[1] or intrinsic PEEP[2] (PEEP$_I$) is an unintentional end-expiratory pressure developed at the alveolar level as a result of incomplete emptying of the lung. It has been estimated that PEEP$_I$ occurs in 39% of mechanically ventilated patients. Mechanically ventilated patients at high risk for developing PEEP$_I$ include those on controlled ventilation receiving minute ventilation of > 10 L, those over 60 years of age, and those intubated for respiratory or cardiopulmonary complications.[3] PEEP$_i$ may occur in patients with or without dynamic airflow limitation (ie, with or without obstructive lung disease) if T_E is too short.

Mechanically ventilated patients, especially those with chronic obstructive pulmonary disease, can have dynamic airflow obstruction leading to PEEP$_I$ because of small-airway collapse. Small-airway collapse, bronchial narrowing (as in asthma), and high minute ventilation lead to dynamic hyperinflation. An inspiratory threshold then develops as a result of PEEP$_I$. During spontaneous effort to trigger the ventilator, the patient must 'pull through' that level of PEEP$_I$ and below the set ventilator-sensitivity level to initiate a breath. This makes triggering the ventilator difficult and increases the work of breathing.

For adequate emptying of the lung to occur, a T_E equal to 3 or 4 respiratory time constants is required (time constant = resistance × compliance). In the normal lung, expiratory driving forces are greater and expiratory resistance is less than in the obstructed lung with increased compliance, providing a lesser respiratory time constant and allowing a shorter T_E. Furthermore, patients may have a limited time for expiration because of high minute ventilation due to burns, sepsis, or large physiologic dead space.[4]

PEEP$_I$ may also develop in relatively normal airways. Components of the patient-ventilator system (the endotracheal tube, PEEP$_e$ valve, and exhalation valve) add resistance and retard expiration. Total expiratory resistance across airways, the endotracheal tube, and the exhalation valve may be > 10 cm $H_2O \cdot s \cdot L^{-1}$ (normal < 4 cm $H_2O \cdot s \cdot L^{-1}$). Thus, PEEP$_i$ can develop in these patients with minute ventilation > 20 L/min.

Patients who actively exhale at end-exhalation have PEEP$_I$ present (without lung distension).[5] Active exhalation from muscle activity results in increased alveolar pressure much like a patient who performs a forced expiratory volume maneuver. The increased alveolar pressure creates a continuous flow that lasts throughout exhalation.

Detection of PEEP$_I$ is difficult. PEEP$_I$ is associated with wheezing and rhonchi as is the presence of flow at end-expiration (the flow curve does not return to baseline before the next breath).[6] During volume-oriented mechanical ventilation, the inspiratory waveform is determined by the ventilator setting. Gas flow is best measured at the Y-piece of the ventilator circuit with a calibrated pneumotachometer of suitable range.[7] PEEP$_I$ is present whenever end-expiratory gas flow does not fall to zero (ie, the pressure difference between alveoli and the airway threshold maintains expiratory flow). Although the presence of PEEP$_I$ can be determined from the expiratory flow tracing, the amount of PEEP$_I$ cannot be determined.

PEEP$_I$ can be measured at the bedside with the patient passive throughout exhalation. Pepe and Marini[1] first described this method, which requires occlusion of the expiratory valve. The time between ventilator cycles is determined, the rate is reduced to zero, and the expiratory valve is occluded when the next breath would have been delivered. If PEEP$_I$ is present, it will be seen on the ventilator manometer when airway pressure equilibrates. Some new-generation ventilators provide the convenience of PEEP$_I$ measurement by an expiratory hold button, but this convenience may be outweighed by the difficulty in obtaining an accurate measurement because of bias flow and the design of the expiration valve.[8] Madsen and colleagues[9] compared PEEP$_I$ determination by the expiratory hold function to clamping-transducing measurement using low-compressible-volume circuits (1.8 mL/cm H_2O) and found no significant difference between the two. It should be noted that the expiratory hold of 4 seconds used in the Madsen et al study[9] requires that a patient be sedated and paralyzed.

Esophageal pressure (P_{es}) must be known to determine PEEP$_I$ in the spontaneously breathing patient, and P_{es} and lung compliance are needed to quantify work of breathing and patient effort during spontaneous breathing. P_{es} provides a reliable method for determining the level of PEEP$_e$ needed to counterbalance PEEP$_i$ in the patient breathing spontaneously during positive pressure ventilation.[10] Knowledge of P$_{es}$ in the patient who is not breathing spontaneously allows computation of the chest-wall compliance and facilitates interpretation of central venous and pulmonary artery pressure.

A challenge to respiratory therapists in the ICU is accurate measurement of static compliance (C$_{st}$) in patients who are being mechanically ventilated. Calculation requires that the change in volume be divided by plateau pressure minus PEEP$_e$. The measurement is accurate if the elastic recoil of the lung minus PEEP$_e$ is zero. In other words, if there is no PEEP$_i$, subtraction of PEEP$_e$ provides a zero baseline and an accurate measurement of C$_{st}$. If PEEP$_i$ is present but not subtracted, then the calculated C$_{st}$ underestimates the correct value.[2,11]

Another critical issue in the presence of PEEP$_e$ is the accuracy of pulmonary capillary wedge pressures (PCWPs). Intrapulmonary vascular pressure measurements are affected by alterations in transmural pressure (the force exerted against the inner surface of the wall of a blood vessel relative to ambient pressure.) Increases in intrapleural pressure decrease vascular transmural pressure and dampen vascular pressure. As a result, actual effective PCWP is equal to the measured PCWP minus the P$_{es}$ measured at end-expiration.[7] In the spontaneously breathing patient, this difference is usually small, but in mechanically ventilated patients, the difference can be appreciable because of the effects of peak airway pressure, PEEP$_e$, inspiratory-expiratory time ratios, and mean airway pressure.

Proper placement of the esophageal balloon is important for accurate readings. In most adults, the tip of the balloon should be about 35-40 cm from the nares.[12] In the spontaneously breathing patient, proper placement can be confirmed by observing P$_{es}$ and airway pressure changes.[13] If the catheter is properly placed, inspiration against an occluded airway results in simultaneous changes in airway pressure and P$_{es}$.[12] Maintaining the patient in an upright position provides the best agreement between pressures; whereas, the poorest position is supine.[1] In the supine patient who is not breathing spontaneously, observation of atrial contractions in the P$_{es}$ trace (Fig. 1) helps assure proper placement.[14] The incorporation of the esophageal balloon into the nasogastric tube facilitates regular measurement of P$_{es}$.

PEEP$_i$ determination with an esophageal balloon requires measurement of P$_{es}$. With inspiration, a slight drop normally occurs before airway pressure and flow increase. Patients with PEEP$_i$ have a greater drop in P$_{es}$ before there is a response in flow or pressure (Fig. 1). The difference between baseline P$_{es}$ and the P$_{es}$ required to generate flow or alter airway pressure is equal to PEEP$_i$.[7]

The techniques used to minimize PEEP are listed in Table 1.[6,8,15] Our patient was receiving aggressive bronchodilator therapy (continuously aerosolized albuterol and I.V. aminophylline), I.V. methylprednisolone, and secretion removal by endotracheal suctioning. Following inspection of the chest radiograph and the monitor study showing dynamic hyperinflation and PEEP$_i$, the inspiratory flow was increased until PEEP$_i$ and hyperinflation were eliminated. PEEP$_i$ increases as T$_E$ decreases. Therefore, increasing inspiratory flow to allow maximal T$_E$ reduces PEEP$_i$. The increased T$_E$ allows more complete alveolar emptying despite dynamic airway compression. However, increased inspiratory flow should be used with caution in patients with bronchospasm, and such patients must be closely monitored. Studies[16-18] have shown that increasing flow can result in increases in P_{aCO_2} and decreases in P_{aO_2} due to maldistribution of inspired gas. Increasing flow to minimize PEEP$_i$ is appropriate as long as ventilation and oxygenation are not sacrificed. If peak inspiratory pressure rises as flow is increased and ventilation is impaired, other techniques for minimizing PEEP$_i$ should be used. In our patient, ventilation and oxygenation remained unchanged and peak pressure rose only from 37 cm H_2O [3.62 kPA] to 41 cm H_2O [4.02 kPA]. Other techniques include reducing minute ventilation consistent with an acceptable pH and attenuating ventilatory drive by treating and correcting fever, agitation, and metabolic acidosis.

Table 1. Clinical Techniques for Minimizing Auto-PEEP

- Decrease airflow obstruction by
  - Aggressive bronchodilation therapy
  - Secretion removal
  - Increasing the size of the endotracheal tube
  - Chest physical therapy
  - Inhaled anesthesia
- Modify ventilatory pattern by
  - Minimizing inspiratory time in relation to total breathing cycle time by
    - Increasing inspiratory flowrate
    - Substituting a low-compressible volume circuit
  - Prolong expiratory time by
    - Decreasing rate
    - Increasing tidal volume
  - Use SIMV at a lower rate
- Apply PEEP/CPAP
- Normalize pH by
  - Administering $NaHCO_3$ during metabolic acidosis
  - Allowing increase in P_{aCO_2} to 50-60 mm Hg range by decreasing rate and normalizing pH.
  - Avoiding purposeful hyperventilation

Adapted from reference 6 and 8 with permission.

In our patient, PEEP$_e$ was not increased above 10 cm H_2O because of the potential for hemodynamic compromise. The hemodynamic consequences of PEEP$_i$ and PEEP$_e$ are the same. Intrathoracic pressure remains positive in the obstructed, compliant lung for a longer period of time, with a consequent rise in mean pressure. The compliant lung transmits alveolar pressure to intrathoracic vessels and, thereby, decreases venous return and cardiac output. Tobin and Lodato[19] point out that

$PEEP_e$ should be used cautiously in patients with airway obstruction. $PEEP_e$ may reduce the work of breathing but does nothing to reduce hyperinflation. Maneuvers that help reduce mean intrathoracic pressure and the hemodynamic effects of $PEEP_i$ are directed toward reducing airflow obstruction.

In Summary

The patient was admitted for severe asthma and bronchitis, with complaints of increased shortness of breath, sinus congestion, and sputum production over a 2-week period. In the emergency room, treatment was begun with aerosolized albuterol, I.V. aminophylline, and oxygen. Over the next 6 hours, the patient deteriorated and required 100% oxygen and I.V. methylprednisolone. Further deterioration lead to intubation and mechanical ventilation. A chest radiograph showed hyperinflation and a waveform study revealed hyperinflation and $PEEP_i$. An increase in ventilator inspiratory flow from 50 to 100 L/min resulted in decreases in $PEEP_i$ and hyperinflation. The inspiratory flow was then increased to 120 L/min, which eliminated hyperinflation and $PEEP_i$ without altering ventilation and oxygenation. This case illustrates the effects of altering inspiratory flow on dynamic hyperinflation and $PEEP_i$ in a mechanically ventilated patient with asthma and bronchitis. The patient responded to aggressive management, mechanical ventilation was discontinued on Day 8, and the patient was subsequently discharged.

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Progressive Respiratory Failure, Toxic Delirium, and Shock in a 47-Year-Old Man

Digpal Chauhan MD and Julie Johnson RRT

Case Summary

History

A 47-year-old divorced, white man was admitted with the chief complaints of cough that was mostly nonproductive and fever and chills for the 2 weeks before admission. Sputum was scant and whitish. The patient had experienced no hemoptysis, pleuritic chest pain, or dyspnea on exertion, but he had sustained a 30-lb weight loss over a 3-month period. He had not experienced headache or sore throat, denied tick bite and recent rash, travel, or the acquisition of new pets. He had lived in Kansas and Missouri for the last few years. Nothing in his history suggested bronchial asthma, pulmonary tuberculosis, or cystic fibrosis. The patient had a smoking index of 60 pack-years but had recently quit. He had abused ethanol in the past but had never used intravenous drugs, required a blood transfusion, or engaged in homosexual acts. He took no medications.

Physical Examination

The patient appeared to be debilitated and chronically ill. His respiratory rate was 28/min, temperature 102° F, height 74.5 in., weight 131.8 lb (ideal weight 195 lb), pulse 112 beats/min, and blood pressure 109/82. There was no periodontitis, adenopathy, digital clubbing, or stiffness of the neck. The patient was alert and oriented to time and place but unable to name the current President or Vice-President. Auscultation revealed a soft S3 heart sound without murmur or friction rub. The electrocardiogram revealed sinus tachycardia, left-axis deviation, and left anterior hemiblock and QS pattern in Leads V1-V3, suggesting an old anteroseptal myocardial infarction. The results of laboratory tests, including arterial blood gas analysis with the patient receiving 4 L/min of oxygen by nasal cannula, are shown in Table 1. The chest radiograph is shown in Figure 1. Following the initial examination and testing, the patient deteriorated rapidly and required intubation and mechanical ventilation.

Table 1. Laboratory Findings in a 47-Year-Old man Presenting with Weight Loss, Nonproductive Cough, and Chills and Fever

Variable	Test Results	Normal Range
Hematocrit	44%	38-49%
WBC*	7,100 cells/mm^3, with 26 band & no lymphocytes	4,000-10,000 cells/mm^3
SGOT	165 IU/L	8-42 IU/L
BUN	22.5 mg/dL	5-10 mg/L
Serum albumin	2.7 g/dL	3.5-4.9 g/dL
Serum creatinine	1.2 mg/L	0.5-1.1 mg/dL
Serum sodium	124 mmol/L	136-144
Serum phosphorus	2.5 mmol/L	2.6-4.9 mmol/L
CPK	elevated	—
CPKI	100% MM	—
pH	7.55	7.36-7.44
Bicarbonate (HCO_3^-)	22.5 mEq/L	24-28 mEq/L
P_{aCO_2}	25 torr†	36-44 torr
P_{aO_2}	70 torr, A-aD_{O_2} = 194 torr	normal A-aD_{O_2} on 4L/min O_2 = 125 torr
S_{aO_2}	96.2 %	—

*WBC = White blood cell count; SGOT = serum glutamic oxalacetic transaminase; BUN = blood urea nitrogen; CPK = creatine phosphokinase; CPKI = creatine phosphokinase isoenzyme; MM = skeletal muscle.

†Indicates acute respiratory alkalsosis.

Dr Chauhan is Chief of Pulmonary, Director of ICU & Respiratory Therapy, and Clinical Assistant Professor of Medicine, Kansas University Medical Center, Kansas City, Kansas. Dr Chauhan is a staff physician and Ms Johnson is supervisor of Respiratory Therapy—Dwight David Eisenhower VA Medical Center, Leavenworth, Kansas.

Reprints & Correspondence: Digpal Chauhan MD, DDE VA Medical Center, 4101 South 4th St, Leavenworth KS 66048

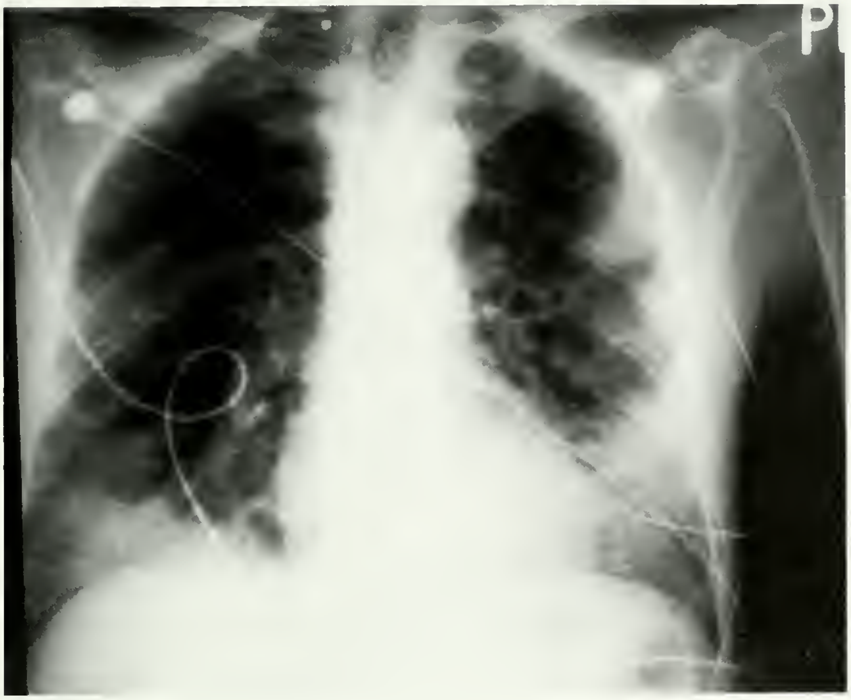

Fig. 1. Chest radiograph of the 47-year-old man at hospital admission.

How would you answer these questions?

What abnormalities are seen in Figure 1?

What diagnoses should be considered, given the history, results of physical examination, and laboratory and radiographic findings?

What other diagnostic procedures and therapeutic actions do you suggest?

Answers and Discussion on Page 302

Radiographic Findings. The chest radiograph reveals hyperinflation and multiple areas of alveolar consolidation in both lower lobes and in the midzone of the left lung field with no evidence of pleural effusion.

Differential Diagnosis. Possible diagnoses to account for the results of testing, examination, and the chest film include pneumococcal pneumonia, viral pneumonia, pneumocystis pneumonia, fungal pneumonia, HIV infection, and lymphoma (lymphomatoid granulomatosis).

Further Tests & Therapy. Positive identification of the causal organism is imperative. Sputum studies, bronchoscopy, and bronchoalveolar lavage may aid the identification.

Clinical Course

The patient's condition worsened rapidly. He was treated initially and empirically for legionella pneumonia with azithromycin and intravenous (I.V.) trimethoprim-sulfamethoxazole. Deterioration continued and the patient became confused, markedly hypoxic, and tachycardic.

In order to establish positive identification of the causal organism, sputum was cultured and stained with direct fluorescent antibody (DFA) and bronchoscopy was performed. Sputum cultures grew only normal flora, but DFA staining was positive for *Legionella pneumophila*. Special stains of bronchial washings were negative for tuberculosis and pneumocystis but grew 8 colonies of *L. pneumophila*. Bronchoscopy revealed tracheobronchitis and frothy secretions. Bronchial washings and viral cultures were also negative. Cerebrospinal fluid (CSF) studies were negative. CD-4 cell count was 169, and the results of human immunodeficiency virus testing were negative. Computerized tomography revealed mild cortical atrophy. Titers for *Legionella spp* during the acute illness were positive at 1:64. The patient did not return for convalescent titers.

The patient required mechanical ventilation for 6 days and exhibited sinus tachycardia with supraventricular tachycardia for the first 3 days. The supraventricular tachycardia responded temporarily to adenosine. Dopamine was required for several days to sustain adequate blood pressure. The patient was treated for 10 days with azithromycin, 14 days with erythromycin (including 7 days of I.V. infusion), 14 days of ciprofloxacin (including 7 days of I.V. infusion), and 7 days of doxycycline. The patient was discharged on Day 17. On subsequent return to the chest clinic, he was found to be doing well and to have regained his weight.

Discussion

This patient sustained septic shock, respiratory failure, myocardial depression, encephalopathy, and rhabdomyolysis (as reflected by the elevated CPK) as a consequence of infection with *L. pneumophila*. A combination of antibiotics was required to control the infection. Questioning revealed the presence of an old air conditioner at his home, which may have been the source of the legionella although surveillance cultures could not be obtained.

Epidemiology

Legionnaire's disease (LD) was first recognized in 1976 in Philadelphia when 34 deaths from pneumonia occurred among the conventioneers. Legionella is ubiquitous worldwide in the water ecosystem[1] and may account for 0.5-5.0% of pneumonia in adults.[2] Natural reservoirs include lakes, ponds, and streams, but potable water sources, particularly plumbing systems in institutions and air conditioning cooling towers, appear to be reservoirs as well. Shower heads, faucets, drinking fountains, whirlpool baths, sprinkler systems, and landscape fountains may be sources as may be room humidifiers and some types of respiratory therapy equipment. Therefore, tap water should not be used for rinsing nebulizers, tubing, or humidifiers.[3] Some cases of nosocomial LD may be due to microaspiration of legionella-contaminated water, associated with nasogastric tube use. The incubation periods for LD is 2-10 days, major risk factors for LD include renal dialysis, renal transplantation, malignancy, immunosuppression, smoking, chronic ethanol abuse, and diabetes.[1]

Microbiology

Legionella spp are weak-staining Gram-negative bacilli that measure from 0.5 to 0.7 μ in width × 2 to 3 μ in length.[1] They are not usually seen on Gram-stained sputum. Special media (buffered charcoal yeast extract) and growth conditions are required for culture. Legionella are slow growing and require 3-5 days for isolation. Within the genus, at least 22 species have been identified, but *L. pneumophila* accounts for more than 95% of cases of LD. In immunocompromised hosts, *L. micdadei* is the usual pathogen and responsible for the Pittsburgh pneumonia.

Clinical Presentation

The clinical syndrome of LD includes nonproductive cough, high-grade fever with multiple rigors, pulse-temperature dissociation, diarrhea, myalgia, confusion and obtundation, hyponatremia, hypophosphatemia, and abnormal liver function.[2] The sputum may be blood-streaked or purulent. Chest radiographs are variable,[4] showing unilateral or bilateral alveolar consolidation or patchy nonsegmental infiltrates. Cavitation and pleural effusions are not uncommon.[2] One feature of LD is the tendency for radiologic progression despite appropriate antibiotics. Radiologic clearing is slow.[5]

Disease Confirmation

Diagnosis is confirmed establishing the presence of antigens, antibodies, or positive cultures. DFA is an excellent method for detection of *L. pneumophila* in sputum, tissue, and body fluids but requires well-qualified laboratory personnel.[2] DNA probe test is almost as accurate as DFA and does not require a high level of technical skill and is preferred in high-volume laboratories.[2] The probe detects infections caused by all *Legionella spp.* The diagnosis can be confirmed by detection of *L. pneumophila* antigen in the urine. This finding is 80-99% sensitive and 99% specific.[2] Antibody estimation can be made from acute and convalescent titers. Positive cultures are 100% specific and can be obtained from sputum and lung tissue. Polymerase chain reaction for legionella DNA looks promising.[2]

Antibiotic Therapy

Although azithromycin is more active against intracellular legionella,[6] high-dose I.V. erythromycin (1 g every 6 h) is still the first-line drug in critically ill patients. Rifampin is a good addition to erythromycin or doxycycline but should not be used alone. Fluoroquinolone is also a good alternative therapy. Some patients will need a combination of all the drugs to salvage them (Table 2), even when drug treatment is begun early in the hospitalization.

ACKNOWLEDGMENTS

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Table 2. Drug Therapy for Legionnaire's Disease

- Macrolides/Azalides
  - erythromycin (± rifampin)
  - azithromycin
  - clarithromycin
- Fluoroquinolones
  - ciprofloxacin
  - ofloxacin
- Rifampin
- Tetracyclines
  - doxycycline
  - minocycline
- Sulfonamide
  - trimethoprim-sulfamethoxazole (± rifampin)

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Amyotrophic Lateral Sclerosis: Diagnosis and Management for the Clinician, JM Belsh and PL Schiffman, editors. Hardcover, 390 pages. Armonk NY: Futura Publishing Company Inc, 1996. $90.

Amyotrophic lateral sclerosis (ALS) is one of the most common neuromuscular diseases; it almost always results in respiratory muscle failure and the need for home mechanical ventilation; and because 40% of home ventilator users have neuromuscular disease, the new book, **Amyotrophic Lateral Sclerosis: Diagnosis and Management for the Clinician** may be of interest to the respiratory community. In general, the book is well written, and the historical information and the chapter on "Notable People with ALS" are of broad interest. The epidemiology, etiology, diagnosis, and neuropsychology sections are 'top notch.' Considering the many new treatment trials and new medications on the market, this book is current.

Concerning rehabilitation, I believe that the augmentative communication section should have been expanded because it is of paramount importance for many tracheostomized home ventilator users. I would have also included information about patient links to environmental control via computer-driven voice synthesizers and printers. Unlike the authors, in my experience, I have not found lower leg bracing to be useful in typical patients with ALS. Likewise, on the contrary to what is written, I have found no evidence that supports strengthening exercises or incentive spirometry as beneficial for these patients. Citing the benefits of exercise for these patients and others with much more slowly progressive diseases seems inappropriate. I did not understand why a patient with a vital capacity of 500 mL would be asked to perform incentive spirometry to a volume of 500 mL when, in fact, the patient can be insufflated to a volume of 2,000 mL. Manually assisted coughing *must* follow a maximal insufflation to be effective when the vital capacity is so low.[1] Despite these concerns, most general rehabilitation topics were presented nicely.

Considering pulmonary assessment, it is rarely necessary to perform full batteries of pulmonary function tests; likewise, initial arterial blood gas samples are rarely warranted. Peak cough flow measurements[2] and end-tidal CO_2 are not even mentioned even though they, along with spirometry and oximetry, are cheaper, noninvasive, more informative, and directly indicative of specific therapeutic interventions.[2] On the other hand, the authors very appropriately discourage the routine sampling of arterial blood gases, and they appropriately ignore polysomnography.

The greatest shortcoming of this book lies in respiratory management. The book fails to differentiate the management of the patient with severe bulbar (throat) muscle weakness from the significant minority of patients with functional bulbar musculature but totally paralyzed peripheral muscles and diaphragm. The importance of the expiratory muscles and the manual and mechanical methods that can be used to assist them is either poorly presented or entirely ignored. One cannot effectively assist a cough with the lungs "empty." A normal cough expels 2.5 L of air. Patients with low vital capacities need deep insufflations before any attempts at assisting cough. Although range-of-motion of the extremity muscles and exercise therapy are amply presented despite the total lack of evidence of their utility in ALS, no consideration is given to providing deep insufflations (range-of-motion to the lungs and chest wall). In my experience this may maintain pulmonary compliance and permit the patient to cough more effectively, raise the voice, and maintain maximum insufflation capacity.

The authors note "Devices such as nasal ventilation and cuirass ventilators may be temporarily effective and prolong life." Thus, noninvasive ventilation is equated with part-time bi-level ventilation and negative pressure methods; full-time ventilatory support is equated with tracheostomy ventilation. Mechanically assisted coughing, mouth piece and lip seal ventilation, the methods that best permit the use of 24-hour ventilatory support as an alternative to tracheotomy, are ignored.[5,6] The authors also note that a strategy should be to increase respiratory muscle strength. This appears ironic since a study by these same editors[3] demonstrated that even mildly affected amyotrophic lateral sclerosis patients respond to a respiratory muscle resistive exercise program with a *decrease* in vital capacity and inspiratory pressures; and, to my knowledge, no one else has demonstrated benefits of exercise for ALS patients.

The authors also fail to consider criteria for placing or removing tracheostomy tubes[2,4] as well as criteria for permitting long-term 24-hour ventilatory support by noninvasive means for ALS patients.[2] They state that the "more chronic use (of nasal bi-level ventilation) often results in skin breakdown and ulcer formation over the bridge of the nose." However, alternatives to nasal bi-level positive pressure do exist (eg, custom-molded nasal interfaces, interfaces that are alternated, and mouth piece or lip seal ventilation). The authors' approach to using part-time bi-level ventilation at inadequate inspiratory-assist ranges via a simple continuous positive airway pressure (CPAP) mask or part-time negative pressure ventilation, rather than the delivery of noninvasive ventilation by portable volume ventilators with an appropriate array of interfaces, limits a patient's options and may mean that the patient develops acute respiratory failure or undergoes tracheotomy. These physical medicine alternatives may keep many of these patients healthy.[5,6] In my experience, the removal of tracheostomy tubes in many ALS ventilator users is helpful. Noninvasive physical medicine alternatives include the use of manual and mechanical assisted coughing instead of bronchodilators and oxygen, bi-level positive pressure at maximum spans, and the use of volume-cycled ventilators at adequate volumes and with a variety of facial interfaces around the clock as needed. Perhaps if the authors and editors become familiar with these methods, they may find that rates of acute respiratory failure are reduced in ALS patients or that the need for tracheotomy is prevented.

In summary, I disagree with the authors' statement "(noninvasive ventilation is necessarily) less effective than tracheostomy with mechanical ventilation" and that an elective tracheotomy should be considered for patients "as soon as VC drops below 1 liter" without considering criteria for tracheotomy.

I believe it is important for the authors to consider ethical and legal issues concerning mechanical ventilation and to inform patients of all their options. An idea unfortunately not even suggested by this book.

John R Bach MD
Professor and Vice-Chairman
Department of Pulmonary Medicine
& Rehabilitation
University of Medicine & Dentistry of
New Jersey
University Hospital
Newark, New Jersey

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Research Design: Qualitative and Quantitative Approaches, by JW Creswell. Softcover, 228 pages. Thousand Oaks CA: Sage Publications, 1994. $19.50.

This book was written to assist students and faculty who are preparing a scholarly journal article, thesis, or dissertation. The book can be used either as a text for upper division students or as a resource for researchers with various levels of expertise. The book is not a detailed methodology text, rather it is designed to assist researchers in making decisions throughout the research process. Therefore, I agree with the author's recommendation that to take full advantage of this book, it is useful to have a basic familiarity with various quantitative and qualitative designs.

The format for each of the 11 chapters is consistent. Principles about designing either a qualitative or quantitative research study are presented, and then these principles are illustrated with specific examples. Each chapter ends with writing exercises and an annotated bibliography of additional readings. The citations are wonderful references to specific and detailed information regarding research design issues. A major strength is the plans and tips for consistent writing from the beginning and through the duration of a research project. The examples are rich and illustrative of key concepts. The author gives attention to the decision making required for designing and implementing a research project.

Chapter 1 addresses the framework for the study and is organized into 4 major sections: the focus of the study, choosing either a qualitative or quantitative paradigm, and considering methods for collection and analysis within each paradigm. The chapter begins with practical, specific, and sound advice on how to focus a study. The author proposes insightful questions to assist in choosing a focus and presents a convincing argument for why researchers should choose a single paradigm—either qualitative or quantitative as they focus their study. (However, later in Chapter 10, the author addresses some advantages and strategies for considering mixed-methodology studies that combine qualitative and quantitative research. I particularly like the written exercises in Chapter 10 because they are excellent activities for graduate students to illustrate their grasp of research design issues for quantitative versus qualitative research.)

The author gives a convincing explanation of the relationship between research paradigm and research methods. This brief and somewhat simplistic explanation is probably sufficient and may in fact be preferable to facilitate progress and understanding by graduate students. Chapter 1 ends with examples of formats for conceptualizing headings and subheadings when writing.

Chapter 2 focuses on the use of literature in research. Differences and similarities in using literature for qualitative and quantitative studies are addressed in a practical and reasonable fashion. In my opinion, the best part of this chapter and perhaps the entire book is the section on preparing a "research map of the literature." The author describes this useful tool for helping a person understand how the proposed study adds to, extends, or replicates research already completed. A useful model for limiting the literature review is also provided. The suggestions on managing the literature review should be welcomed by the expert as well as the novice researcher.

Chapters 3-5 deal with the 'how-tos' for writing the introduction. These chapters, like most of the book, are written in a straightforward and practical manner. The author elaborates on how difficult it is to write the introduction and the major role of the introduction to set the stage for the entire study. The introduction must create reader interest, establish the problem that leads to the study, place the study in the larger context of scholarly literature, and reach a specific audience. The author gets to the heart of the most frequent questions and problems encountered and how to go about handling the writing and decision making for the background of the problem, the purpose statement, the research questions, and the hypotheses.

Chapter 6 focuses on the use of theory in the design of a research study. This chapter should be most useful to graduate students who are writing a thesis or dissertation. However, the chapter can also be helpful to more experienced researchers who are writing articles for publication because it elaborates how theory should be incorporated into research design and scholarly writing. Chapter 7 deals with issues related to definitions, limitations, and the significance of a study.

Chapters 8-10 address how to design and to write the methods sections of a research study. Chapter 8 deals specifically with quantitative research, and Chapter 9 deals specifically with qualitative research. Chapter 10 does a nice job of elaborating on the usefulness and difficulties with mixed-methodology designs.

The last chapter provides a general discussion on scholarly writing. 'Macrolevel' writing strategies and 'microlevel' applications are provided. This is a great chapter for anyone who has writer's block or difficulty beginning and sustaining the necessary writing. The last chapter artfully demonstrates the intimate relationship between doing research and writing research. It appears that the author took painstaking efforts to make his thoughts explicit and to share ideas that have worked well for him. My favorite line in the book is "all experienced writers know that writing is thinking and conceptualising a thought" (Page 194).

I believe this book is a useful tool for improving the thinking, performing, and writing of research. The book is ideal for graduate students who have completed courses in research design methods and are beginning their academic and research careers. The book is also recommended for graduate student advisors who are preparing students to plan and to write independent research studies. Finally, the book is an excellent primer for researchers who are experienced with the scientific process of quantitative research but are generally unfamiliar with qualitative research.

Shelley C Mishoe PhD RRT
Professor, Schools of
Allied Health Sciences
Graduate Studies Chair, Department of
Respiratory Therapy
Medical College of Georgia
Augusta, Georgia

Life Support: A Family Clinical Guide, by MP Hahn. Illustrated. 305 pages. Jefferson, North Carolina: McFarland & Company, 1996. $29.50.

The best place to put this reference is in a visible area of an intensive care unit (ICU) waiting room or with other reference books for each allied health and nursing department. This reference may be of use to families or novice health practitioners who are trying to understand the 'high-tech' therapies and interventions (eg, extracorporeal membrane oxygenation or mechanical ventilation) a loved one or patient is receiving in the ICU.

The intended readers for this book are lay persons (who may find the reading level demanding due to the nature of the content) and health care personnel not normally exposed to life support systems. The author focuses on clinical therapeutics, although physiology and pathophysiology are briefly discussed. The first 5 chapters cover topics relating to nutrition, hydration, circulation, and breathing during 'normal' conditions. In addition, the author presents the various clinical and ICU interventions that may be necessary to sustain life when these functions go awry. The last chapter relates to neuroscience and includes important discussion about the neurologic evaluation for the determination of brain death and persistent vegetative state. Readers of this reference may have a better perspective about these matters when or if a crucial decision must be made regarding do-not-resuscitate orders, the withdrawal of life support, and the evaluation of quality of life, terminal disease, and terminal illness. In the text, the author uses a style that may stimulate thoughts and questions in the mind of the reader.

That the most comprehensive chapter is on ventilatory support comes as no surprise, because the author is a respiratory care practitioner. Important discussion in this chapter centers around ventilator alarms, chest tubes, and the possible administration of narcotics for the relief of dyspnea and pain. In addition, the author discusses the phenomenon in some patients known as 'fighting the ventilator.'

With one exception, the content is accurate, with helpful illustrations. The units for systemic vascular resistance and pulmonary vascular resistance are in error, as is commonly the case in other references. These units are stated as dynes/sec/cm^{-5}, rather than as dyne · s · cm^{-5}.

Although I have more than 20 years of ICU experience, I found that some of content in this book is new, interesting, and applicable. I intend to keep it handy.

Keith R Momii MMSc MA RRT RPFT
Department of Pulmonary Care Services
The University of Texas Medical
Branch at Galveston
Galveston, Texas

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RETROSPECTROSCOPE

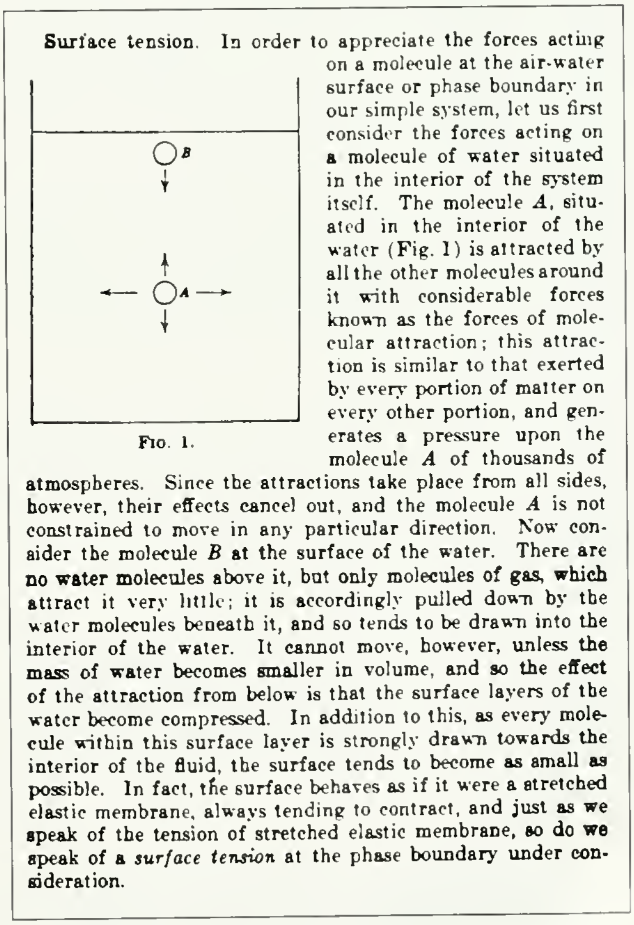
Premature Science and Immature Lungs
Part 1: Some Premature Discoveries

When I was a medical student, we studied the tenth edition of Holt's *Diseases of Infancy and Children.* I still have my copy, 45 years later. The tenth edition, as were its predecessors, was authoritative and up to date; published in 1932, it contains references to many articles published in 1931 or 1932. But among its 1,240 pages there is no mention of hyaline membrane disease or respiratory distress syndrome. There are three pages on asphyxia of the newly born and another three on congenital atelectasis; when these conditions occurred in premature babies, the authors thought it likely that they were due to lack of development of the respiratory center.

Three years earlier, a Swiss respiratory physiologist, Kurt von Neergaard, had published a now-classic paper (1); its title (translated from German into English is "New notions on a fundamental principle of respiratory mechanics: the retractile force of the lung, dependent on the surface tension in the alveoli." Much more about Neergaard later, but in that 1929 paper he demonstrated clearly that surface forces at the interface of moist alveolar walls and alveolar gas caused a strong retractile force (even stronger than that of stretched elastic tissues) tending to collapse alveoli and he speculated that atelectasis of the newborn might be due to the considerable retractile force of surface tension that opposes the first expansion of the lungs.

It's too much to expect that the two pediatricians responsible for the tenth edition of Holt's text had noticed and read Neergaard's 1929 article in a German journal of experimental medicine in time to have included it in their 1932 revision, so I looked in the medical physiology texts of the 1930s, then those of the 1940s and then those of the 1950s for a discussion of surface forces. They contained little or nothing on surface tension and nothing at all on a possible role that surface tension might play in expansion and retraction of the lungs.

What is surface tension? Figure 1 explains it well. Possibly there are better definitions but I have selected this one because it keeps me humble; it comes from pages 2 and 3 of the text (2) that I used as a second-year college student in 1929 in L. V. Heilbrunn's stimulating course in general physiology at the University of Pennsylvania! Indeed, all of Chapter 1 (24 pages) was devoted to surface action and ways of measuring surface tension. Much later in life, I became a pulmonary physiologist and was even associated briefly with a pediatrician who measured pressure-volume curves of atelectatic lungs from babies who had died from hyaline membrane disease.



Surface tension. In order to appreciate the forces acting on a molecule at the air-water surface or phase boundary in our simple system, let us first consider the forces acting on a molecule of water situated in the interior of the system itself. The molecule *A*, situated in the interior of the water (Fig. 1) is attracted by all the other molecules around it with considerable forces known as the forces of molecular attraction; this attraction is similar to that exerted by every portion of matter on every other portion, and generates a pressure upon the molecule *A* of thousands of atmospheres. Since the attractions take place from all sides, however, their effects cancel out, and the molecule *A* is not constrained to move in any particular direction. Now consider the molecule *B* at the surface of the water. There are no water molecules above it, but only molecules of gas, which attract it very little; it is accordingly pulled down by the water molecules beneath it, and so tends to be drawn into the interior of the water. It cannot move, however, unless the mass of water becomes smaller in volume, and so the effect of the attraction from below is that the surface layers of the water become compressed. In addition to this, as every molecule within this surface layer is strongly drawn towards the interior of the fluid, the surface tends to become as small as possible. In fact, the surface behaves as if it were a stretched elastic membrane, always tending to contract, and just as we speak of the tension of stretched elastic membrane, so do we speak of a *surface tension* at the phase boundary under consideration.

Fig. 1.

Fig. 1. Definition of surface tension by Eric Ponder (2).

Part 1 is reprinted with permission of the American Lung Association, from Am Rev Respir Dis 1977;116:127-135.

But I had total *lack* of recall of Ponder's Chapter 1 and the thought never occurred to me that forces at an air-liquid interface might be keeping alveoli closed. I had lots of very good company in failing to make this association and in failing to appreciate a few small voices that did make it. Fortunately, a series of events in the mid- and late 1950s led to the discovery of pulmonary surfactant, a unique substance formed and secreted by pulmonary Type II alveolar cells, that decreases surface tension to very low values at low alveolar volume and so serves as an anti-collapse factor. Absence of this surfactant (as in the lungs of some newborn babies, particularly premature ones) favors collapse of the lungs and puts an almost impossible burden on the weak inspiratory muscles of newly born premature babies. Because the key to understanding, to diagnosing before birth, and to managing respiratory distress syndrome before and after birth is a knowledge of surface tension and the behavior of surface films, let's start at the beginning—with the discovery of surface tension.

Pierre Simon Laplace and Surface Forces

The Marquis de Laplace, sometimes called a latter-day Newton, was a brilliant French mathematician, physicist, and astronomer. His genius was recognized early and he became professor of mathematics in the École Militaire de Paris. One of his students there was Napoleon Bonaparte. Certainly Laplace benefitted from this association, because Napoleon richly rewarded Laplace with estates and a Ministry, forgave him his administrative ineptitudes, and continued to favor Laplace during Napoleon's 100 days' return to France (even though Laplace was among those who had signed the earlier order banishing Napoleon). Laplace had an amazing talent not only for mathematics but also for political survival, for although his friend Lavoisier went to the guillotine during the Reign of Terror in 1794, Laplace not only kept his head but his estates and wealth as well.

Laplace devoted his scientific career to a profound theoretical analysis of the solar system and between 1798 and 1827 published five huge tomes—more than 2,000 pages in all—entitled *Traité de Mécanique Céleste* (3). It dealt with the general laws of mechanics, gravitation, and motions of celestial bodies. Certainly it was a sequel to Newton's *Principia* and some rated it as intellectually on par with it.

What does Laplace's study of the heavenly bodies have to do with surface forces? In Tome IV of *Mécanique Céleste* are two supplements on "La théorie de l'action capillaire"—read before the French Academy and published in 1806 and 1807. On page 689 of Volume IV of Bowditch's translation (4) is Laplace's presentation of his general law in words:

> From these results, relative to bodies terminated by sensible segments of a spherical surface, I have deduced this general theorem. "*In all the laws which render the attraction insensible at sensible distances, the action of a body terminated by a curve surface, upon an infinitely narrow interior canal, which is perpendicular to that surface, at any point whatever, is equal to the half sum of the actions upon the same canal, of two spheres which have the same radii as the greatest and the least radii of curvature of the surface at that point.*"

On page 823 is Laplace's equation (figure 2). It contains his formulation of the relationship between force, surface tension, and radii of curvature of surfaces.

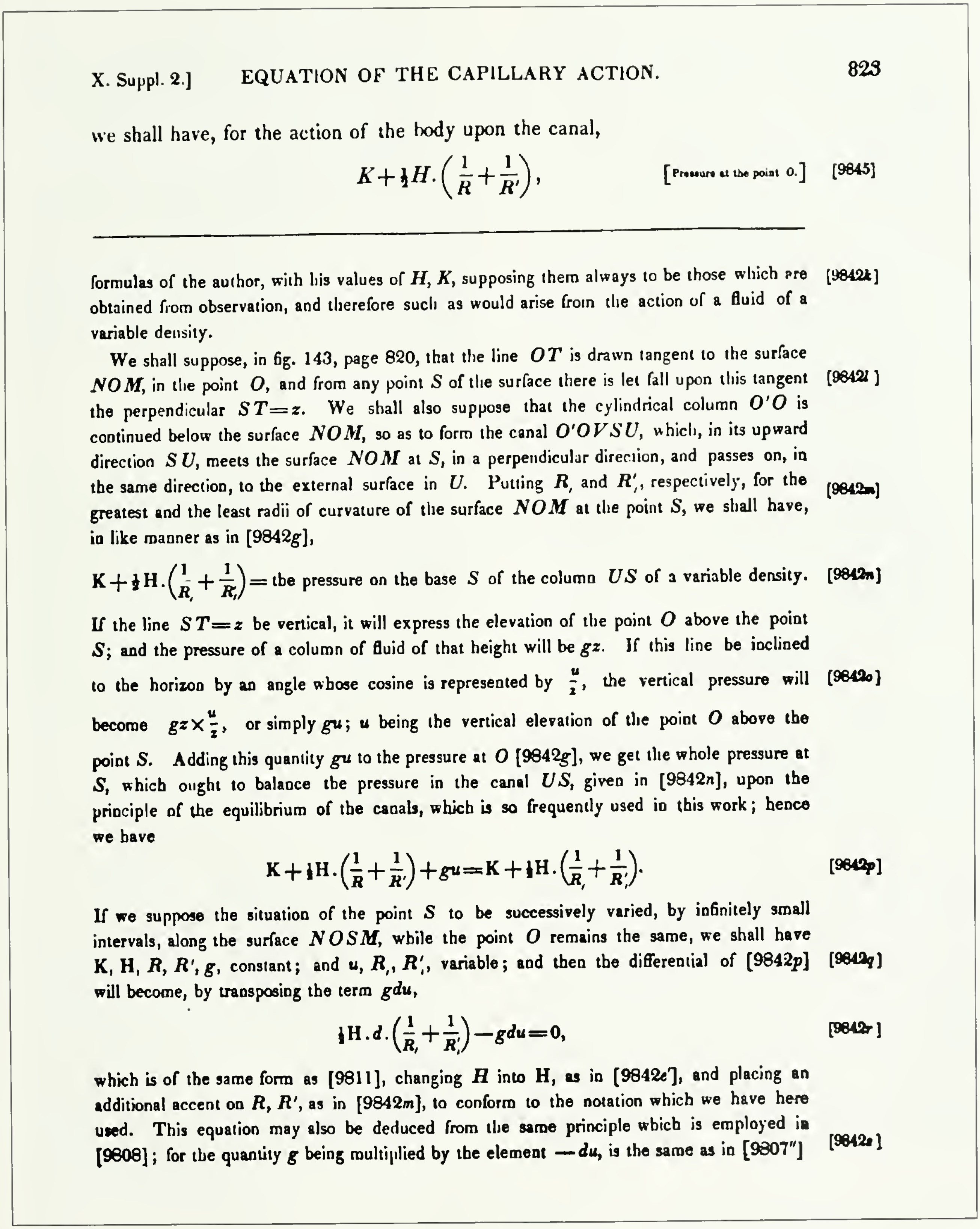
Two question call for answers: (1) *What does capillary action have to do with surface tension?*

One of the simplest and earliest methods of measuring (statically) the surface tension of fluids of biological interest was to observe the height to which a column of fluid rises in a capillary tube. Actually, this was one of the methods used by Neergaard in 1929. A tube with an internal diameter of about 0.5 mm is dipped into the liquid: provided the liquid "wets" the internal wall of the tube, the meniscus rises above the flat surface of the water outside the tube, and it is pulled upward by surface forces (that tend to contract the surface until balanced by the downward force of gravity on the column of liquid. Laplace (see p. 1,009 of Volume IV of Bowditch's English translation) gives his explanation of why fluid rises in a capillary tube above its expected level:

> At the surface of a fluid, the attraction of the particles, modified by the curvature of the surface and of the sides of the vessel which contains it, produces the capillary attraction. Therefore these phenomena, and all those which chemistry presents, correspond to one and the same law, of which now there can be no doubt. Some philosophers have attributed the capillary phenomena to the adhesion of the fluid particles, either to each other, or to the sides of the vessels which contain them; but this cause is not sufficient to produce the effect. For, if we suppose the surface of water, contained in a glass tube, to be horizontal, and upon a level with that of the water in the vessel into which the tube is dipped by its lower end; the tenacity of the fluid, and its adhesion to the tube, would not curve this surface and render it concave. To produce that, it is necessary to suppose that there is an attraction in the upper part of the tube, which is not immediately in contact with the fluid.

(2) *Why do two supplements dealing with capillary action (and surface forces) belong in a treatise on the movements of planets and stars?* Let Henri Poincaré (5) answer that:

> The astronomic universe is formed of masses, very great, no doubt, but separated by intervals so immense that they appear to us only as material points. These points attract each other inversely as the square of the distance, and this attraction is the sole force which influ-



X. Suppl. 2.] EQUATION OF THE CAPILLARY ACTION. 823

we shall have, for the action of the body upon the canal,

$$K+\tfrac{1}{2}H.\left(\frac{1}{R}+\frac{1}{R'}\right),$$ [Pressure at the point O.] [9845]

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formulas of the author, with his values of H, K, supposing them always to be those which are obtained from observation, and therefore such as would arise from the action of a fluid of a variable density. [9842k]

We shall suppose, in fig. 143, page 820, that the line OT is drawn tangent to the surface NOM, in the point O, and from any point S of the surface there is let fall upon this tangent the perpendicular $ST=z$. [9842l] We shall also suppose that the cylindrical column $O'O$ is continued below the surface NOM, so as to form the canal $O'OVSU$, which, in its upward direction SU, meets the surface NOM at S, in a perpendicular direction, and passes on, in the same direction, to the external surface in U. Putting $R_,$ and $R'_,$, respectively, for the greatest and the least radii of curvature of the surface NOM at the point S, we shall have, in like manner as in [9842*g*], [9842m]

$$\mathrm{K}+\tfrac{1}{2}\mathrm{H}.\left(\frac{1}{R_,}+\frac{1}{R'_,}\right)= \text{the pressure on the base } S \text{ of the column } US \text{ of a variable density.}$$ [9842n]

If the line $ST=z$ be vertical, it will express the elevation of the point O above the point S; and the pressure of a column of fluid of that height will be gz. If this line be inclined to the horizon by an angle whose cosine is represented by $\frac{u}{z}$, the vertical pressure will become $gz\times\frac{u}{z}$, or simply gu; u being the vertical elevation of the point O above the point S. [9842o] Adding this quantity gu to the pressure at O [9842*g*], we get the whole pressure at S, which ought to balance the pressure in the canal US, given in [9842*n*], upon the principle of the equilibrium of the canals, which is so frequently used in this work; hence we have

$$\mathrm{K}+\tfrac{1}{2}\mathrm{H}.\left(\frac{1}{R}+\frac{1}{R'}\right)+gu=\mathrm{K}+\tfrac{1}{2}\mathrm{H}.\left(\frac{1}{R_,}+\frac{1}{R'_,}\right).$$ [9842p]

If we suppose the situation of the point S to be successively varied, by infinitely small intervals, along the surface $NOSM$, while the point O remains the same, we shall have K, H, R, R', g, constant; and u, $R_,$, $R'_,$, variable; and then the differential of [9842*p*] will become, by transposing the term gdu, [9842q]

$$\tfrac{1}{2}\mathrm{H}.d.\left(\frac{1}{R_,}+\frac{1}{R'_,}\right)-gdu=0,$$ [9842r]

which is of the same form as [9811], changing H into H, as in [9842*e*], and placing an additional accent on R, R', as in [9842*m*], to conform to the notation which we have here used. This equation may also be deduced from the same principle which is employed in [9808]; for the quantity g being multiplied by the element $-du$, is the same as in [9807″] [9842s]

Fig. 2. Laplace's law. This states that force is proportional to the mathematical expression in line 1, in which K = "intrinsic pressure," H = surface tension, and R and R' are the radii of curvature. Figure 2 in p. 823 of Volume IV of Bowditch's translation (4). Laplace's contribution to p. 823 consists of only the first line; the remainder of the page is filled with Bowditch's annotation and explanation of the preceding page.

> ences their movements. But if our senses were sufficiently keen to show us all the details of the bodies which the physicist studies, the spectacle thus disclosed would scarcely differ from the one the astronomer contemplates. There also we should see material points, separated from one another by intervals, enormous in comparison with their dimensions, and describing orbits according to regular laws. These infinitesimal stars are the atoms. Like the stars proper, they attract or repel each other, and this attraction or this repulsion, following the straight line which joins them, depends only on the distance. The law according to which this force varies as function of the distance is perhaps not the law of Newton, but it is an analogous law; in place of the exponent—2, we have probably a different exponent, and it is from this change of exponent that arises all the diversity of physical phenomena, the variety of qualities and of sensations, all the world, colored and sonorous, which surrounds us; in a word, all nature.
>
> Such is the primitive conception in all its purity. It only remains to seek in the different cases what value should be given to this exponent in order to explain all the facts. It is on this model that Laplace, for example, constructed his beautiful theory of capillarity; he regards it only as a particular case of attraction, or, as he says, of universal gravitation, and no one is astonished to find it in the middle of one of the five volumes of the Mécanique Céleste.

Nathaniel Bowditch

Laplace's *Mécanique Céleste* came to the attention of English-speaking scientists largely through Bowditch's translation (4). Bowditch in some ways is more remarkable than Laplace himself. Bowditch, an American with no formal schooling beyond the age of 10, signed on as a clerk on a sailing vessel in 1795 at the age of 22 and was at sea for most of the next 9 years. During that time, he made his own studies of navigation that resulted in his correcting more than 8,000 errors in the then-bible of navigators, Moore's *Practical Navigator*. His great interest in the planets and stars led him to Laplace's *Mécanique Céleste* and he devoted more than 30 years of his life to translating it into English. He never completed Tome V, but his translation of the first four Tomes (10 "Books") occupies more than 3,700 pages.* More remarkable than his translation are his annotations and explanations of Laplace's work and his corrections of Laplace's many errors. His annotations were helpful even to expert mathematicians because Laplace's mathematics was highly sophisticated and difficult to follow; Bowditch's corrections were essential because previously Laplace's errors went unnoticed. Figure 2 shows the extent of Bowditch's contributions to Laplace's work; of Bowditch's 3,700 pages, about half are a translation of Laplace's French edition and the remaining half are Bowditch's annotations. All of this from an unschooled young man in a rough new world.†

Bowditch had other accomplishments. A son, Henry Ingersoll Bowditch, was a professor of clinical medicine at Harvard (1859-1867) and an early specialist in diseases of the chest. A grandson, Henry Pickering Bowditch, who established the first physiological laboratory in America (1871), was one of the three founders of the American Physiological Society, a co-editor of the new *American Journal of Physiology*, first president of the American Physiological Society, and a Dean of the Harvard Medical Faculty.

Thomas Young

As so often happens in science, the first was not the first. Laplace read his paper to the Institute of France in 1805 and it was published in 1806 as a supplement to *Mécanique Céleste*. Young read *his* paper, "An essay on the cohesion of fluids," before the Royal Society of London December 20, 1804, and it was published in the Philosophical Transactions for 1805 (6). Young's paper contained, in words, most of what Laplace presented a year later in elaborate, formidable, and impressive mathematical terms. Notorious for borrowing from the work of others (and giving little or no credit to them), Laplace made no reference to Young in his first supplement and discussed Young's work in one brief paragraph in his second supplement a year later. Young, in some "additional remarks" added to an 1807 reprint of his 1805 article (6), took exception to Laplace's behavior in the following nicely composed paragraph.

> In an essay read to the Institute of France in December 1805, and published in 1806, as a supplement to the Mécanique Céleste, Mr. Laplace has advanced a theory of capillary attraction, which has led him to results nearly similar to many of those which are contained in this paper [Young's 1805 paper]. The coincidence is indeed in some respects so striking, that it is natural, upon first impression, to inquire whether Mr. Laplace may not be supposed either to have seen this essay, or to have read an account of its contents in some periodical publication; but upon further reflection, we cannot for a moment imagine a person so high and so deserved a reputation as Mr. Laplace, to wish to appropriate to himself any part of the labors of others. The path which he has followed is also extremely differ-

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* Only 500 copies of Bowditch's original translation were printed, and most of these are held in "rare books" collections, but the Chelsea Publishing Company, Bronx, New York, reprinted the four volumes in 1966 ($125 per set in 1976).

†For a 168-page biography of Bowditch, written by his son Nathaniel, see Volume IV of Bowditch's translation (4) or Volume I of Chelsea Company's reprint (4).

> ent from that which I had taken; several of the subjects which I had considered as belonging to the discussion, have not occurred to Mr. Laplace; and it is much more flattering than surprising, that, to an assembly of philosophers not extremely anxious to attend to the pursuits of their contemporaries, investigations should be communicated, by the most distinguished of their members, as new and important, which had been presented, a year before, to similar society in this country.

S. M. Tenney ("The Tangled Web," unpublished manuscript) has proposed that the name of Laplace's law be changed to the "Young-Laplace law." Whether it ever will be or not, Young's fame is secure without it. While a medical student, he established the mechanism by which the lens of the eye accommodates for far and near vision. He later formulated a theory of color vision (the Young-Helmholtz theory), he discovered the principle of interference of light, proposed the wave theory of light transmission, contributed to the theory of tides, and established a coefficient of elasticity (Young's modulus). Most fascinating of all his accomplishments was his contribution to deciphering the Rosetta stone. Young provided the important clue necessary for unlocking the Egyptian system of writing; it was his idea that, in the transliteration of non-Egyptian names, hieroglyphic symbols with phonetic values would be used. The credit for the full translation in 1822 goes to Champollion the Younger, but Young's 1819 article persuaded Champollion to drop his conviction that hieroglyphic symbols were never phonetic.

The Long Quiet Period

It took almost 100 years after Bowditch's translation for Laplace's law to become of interest to biomedical scientists. Neergaard's work on the lung came in 1929. And Burton, who applied Laplace's law to the stability of small blood vessels in 1951 (7), stated that the only previous application he found to a vascular problem was that of Woods in 1892 (8). Woods, a throat surgeon at the Richmond Hospital in Dublin demonstrated that the pressure in the cardiac ventricles depended not only on the tension developed by the contracting muscle but also on the size and shape of the heart (i.e., the principal radii of curvature).** In 1960 Burton devoted six pages of his chapter in Ruch and Fulton's textbook of medical physiology (9) to applications of Laplace's law to the heart and vessels; the same book contained no mention of any role of surface tension in the lungs, although Neergaard's 1929 experiments had been discovered, repeated, confirmed, and published in 1954 by Radford (10).

However, the long quiet period in which mammalian physiologists and biophysicists failed to become interested in surface forces was a very busy one for physicists and chemists. Lord Rayleigh, 1904 Nobel laureate in physics, used surface physics to prepare monomolecular films of liquids and to determine the dimensions of molecules (11). Langmuir, working in General Electric's Research Laboratory, developed a more precise method in 1917 (12) for measuring molecular dimensions and orientation using technics of surface physics and chemistry. He must have had his eye on a Nobel prize in chemistry (which he won in 1932) because he devoted two and a half pages of fine print at the beginning of his 1917 article to establishing in great detail his priority in the experiments he was about to report; he opened with the statement that, "The fundamental idea of the orientation of group molecules in the surfaces and in the interior of liquids as a factor of vital importance in surface tension and related phenomena occurred to me in nearly its present form in June and July, 1916."

Other physicists developed methods that were suitable for measuring surface tension under dynamic conditions; these were eventually used by biologists. Maxwell devised his frame (13), Whilhelmy his surface balance (14), Langmuir his balance (12), and Adam his improved Langmuir balance (15). And Ms. Agnes Pockels insensitively referred to as *Miss* Agnes Pockels by Lord Rayleigh) "described a simple method for increasing or diminishing the surface of a liquid in any proportion, by which its purity may be altered at pleasure" (16). Ms. Pockels apologized for her boldness, not being a professional physicist, in writing about her experiments to Lord Rayleigh, who promptly had her paper published in *Nature*.

And beginning in the 1920s, numerous monographs and reviews appeared on interfacial phenomena, surface chemistry, soap films, foams, surface physics, and monomolecular layers; a World Congress on Surface Active Agents was held in 1954.

These subjects were also of great interest to general and cellular physiologists and especially to those interested in membranes. William Bayliss' first edition of *Principles of General Physiology*, published in 1915, devoted Chapter III to surface action (17). J. B. Leathes, who gave the Croonian Lectures in 1923 (18) on "Role of Fats in Vital Phenomena," described experiments on compressing and enlarging surface films of phospholipids (lecithin) and palmitic acid using Adam's surface balance. Leathes was coming very close to what we now call pulmonary surfactant.

But the mammalian physiologists didn't become interested in alveolar surface tension until 1929, and then only one did—Neergaard.

Back to Neergaard

Neergaard was born in Schleswig-Holstein in Germany. Because he was in poor health as a child, his mother brought him to Davos, the famous Swiss health spa. He remained in

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**Woods also applied Laplace's law to the urinary bladder, uterus, and to arteries and veins.

Switzerland, became a Swiss citizen, and was graduated from the University of Zurich in 1917. In 1922 he moved to Basel where he worked with a well-known internist, Professor Staehelin. But he spent little of his time in the medical clinic in Basel and most of it with physiologists. There he met Dr. Karl Wirz. The Swiss physiologists had been unusually active and innovative in the study of pulmonary mechanics. Rohrer had done his now-classic study on airway resistance in 1915 (19), and Fleisch had devised an excellent apparatus for continuous measurement of inspired and expired air flow in 1925 (20). Wirz, a pupil of Rohrer's, had measured changes in pleural pressure during respiration in 1923 (21). Neergaard joined forces with Wirz to study first lung elasticity, and then flow resistance in air passages of normal humans and those with asthma or emphysema (22, 23).

Shortly thereafter, Neergaard had a novel idea about lung elasticity that had eluded all previous physiologists. He said (1):

> The lung is probably unique among tissues in its extraordinary ability to expand and retract. So far, this has been regarded as due to the elasticity of the tissue itself, and particularly that of elastic fibers. Although attempts have recently been made to determine the elasticity of the elastic fibers directly with the aid of a micromanipulator (Redenz), this does not prove that the entire retractility of lung can be attributed to these structures....
>
> So far, one force has not been taken into account that definitely merits consideration in this context. This is *surface tension*. It is active at the boundary between alveolar epithelium and alveolar air. Wherever the surface of an aqueous solution touches upon a gaseous space, the influence of molecular attraction on the marginal layer of molecules gives rise to tensions in the solution that are described as surface tension. All the phenomena of capillary chemistry, the rise of liquids in fine capillaries, the pressure of soap bubbles, and numerous other phenomena, are based on surface tension. Considerable forces are involved which also play a role in other aspects of respiratory mechanics (as previously pointed out in the evaluation of the so-called adhesion of the pleural surface). The surface tension partly depends on the nature of the solution that constitutes the bordering medium and particularly on the curvature of the boundary. The smaller the radius of curvature, the stronger the force of surface tension, which is like a taut rubber membrane attempting to retract like a rubber balloon. Such deeply curved boundaries between fluid or an aqueous gel (the cellular membrane of the alveolar epithelium) and a gaseous space exist in countless numbers in the pulmonary alveoli....
>
> To visualize the mechanism even more clearly, one should imagine a thin capillary tube at the end of which a soap bubble has been blown. As soon as blowing is discontinued and the inside of the soap bubble is allowed to communicate with the atmosphere via the capillary, the bubble immediately becomes smaller and retracts owing to the influence of surface tension. The smaller the diameter of the capillary in this situation and the smaller the radius of curvature of the bubble, the more rapid and more energetic the retraction. Such small bubbles, which communicate with the atmosphere via narrow capillaries, can be compared to the alveoli of a lung.
>
> In view of the almost complete saturation of expired air with water vapor, we can imagine the alveolar epithelium as covered by a thin film of fluid. The surface tension attempts to make the alveoli smaller, and therefore exerts an influence in the same direction as the retractive force of the entire lung. There can be no doubt, therefore, about its *qualitative* significance, but the quantitative aspects are not known.

Neergaard then set out to obtain quantitative values for pulmonary surface tension. He reasoned as follows:

> To measure the influence of surface tension on retractility, the total retractile force must first be measured by known methods; that is, the so-called elasticity curve in relation to lung volume must be obtained. Since it is impossible to measure surface tension by itself independent of tissue elasticity, our only alternative was to eliminate surface tension in a second series of determinations, thus measuring tissue elasticity separately. The difference between total retraction and the retraction remaining after elimination of surface tension is a measure of surface tension at the same lung volume.
>
> To eliminate surface tension, the lungs had to be filled with a fluid down to the alveoli to remove the effect of the air-tissue interface. ... *the lung was finally emptied of air by a pressure difference method and then immediately filled with an isotonic gum solution.* By the addition of about 7 percent gum arabic to the Tyrode solution, the oncotic pressure was adjusted so that no edema occurred. The solution thus obtained met physiological requirements to a very high degree [see figure 3].

Neergaard divided each experiment into two parts. First he measured the pressure-volume curve with the lung air-filled, and then again with the lung fluid-filled, to eliminate the air-fluid interface and the effects of surface tension; the second curve must then represent the force exerted by elastic tissues. By subtraction, he calculated the force exerted by surface tension. He found, in all states of expansion, that surface tension was responsible for more of total lung retraction then was tissue elasticity. To put it in another way, the pressure needed to distend the lung to a given volume was always higher when the lungs were air-filled and always lower when they were saline-filled. Thus Neergaard, using a direct, simple, unequivocal method, found that the Laplace (or the Young-Laplace) law held for pulmonary alveoli and that surface effects were important forces to be reckoned with in inspiration and expiration.

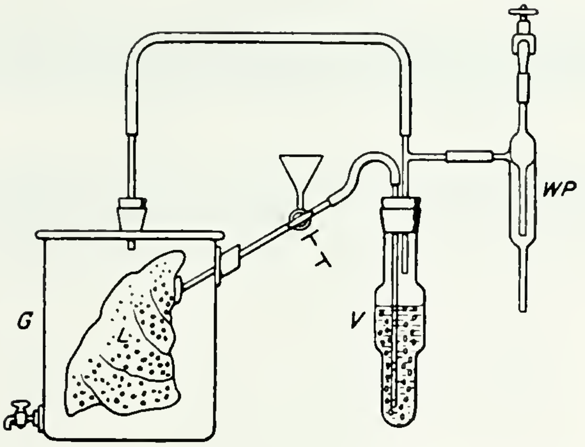

Fig. 3. Apparatus for evacuation of air from the lung. G = glass vessel; L = lung, the main bronchus of which has an airtight connection with the lateral support; V = water valve to obtain a difference between intrapleural and extrapleural pressure; WP = water jet pump. Between WP and V, a mercury manometer is used to determine total retraction on filling with air. From Neergaard (1).

He went further in his experiments, reasoning, and speculation. He reasoned that the surface tension of alveoli may be lower than that of other body fluids, by the accumulation of surface-active substances, according to the Gibbs-Thomson law, and that "it is also conceivable that this [lower tension] would be useful for the respiratory mechanism because without it pulmonary retraction might become so great as to interfere with the adequate expansion." He tried to measure surface tension of aqueous extracts of lungs and found unexpectedly low values of 35 to 41 dynes (water was 73, serum 60-65, and extracts of muscle, spleen, liver, and heart were between 47 and 53 dynes). He was not satisfied with his methods[††] and suggested that the question (of surface tension as a force counteracting the first breath of the newly born) be investigated further. Unfortunately, he himself did not do so, and no one repeated or extended his experiments for more than 25 years.

In 1929, the year in which his now-famous report was published, he moved from Basel back to Zurich to become head physician at the Institute of Physical Therapy there. He never wrote another paper on the lung and did no further scientific investigations. Instead he immersed himself in subjects such as the interrelationship of medicine with philosophy, politics, and social problems (again, ahead of his time). In 1940 he was appointed Director of his clinic and Professor of Physical Therapy in Zurich. He died in 1947, at least a decade before Clements demonstrated clearly, by direct measurement, that lungs contain a unique surfactant and a dozen years before Avery and Mead published direct evidence linking its absence to the respiratory distress syndrome of the newly born.

Why did Neergaard leave Basel and research shortly after making his most important discovery? No on knows for sure. He must have known that it was important because he used the word "Grundbegriff" (fundamental or basic principle) in the title of his article. But he received no recognition for his discovery, then or later in his life. None of his obituaries in 1947 even mentioned that he had ever done research on the lung (just as Paul Bert's obituary told of his wok on skin grafting but said nothing of his momentous study of the effects of high and low atmospheric pressures on man). Dr. Joan Gil has learned that Professor Böni, who was Neergaard's disciple, daily collaborator for his last 18 years in Zurich, and finally his successor, never heard Neergaard mention his work in Basel on pulmonary physiology or express any particular interest in the lung.

Apparently, Neergaard's work fell into the category described by Gunther Stent in his article "Prematurity and Uniqueness in Scientific Discovery" (26); according to Stent; a discovery is premature if its implications cannot be connected by a series of simple logical steps to canonical, or generally acceptable, knowledge. Von Neergaard by temperament was neither a fighter nor a scientific entrepreneur. As a result, his work, which could have led to saving lives of premature babies in the 1930s, suffered a premature death.

Neergaard's work ends Part I of this story. Part II (for the most part) begins 25 years post-Neergaard—in the mid-1950s.

Julius H. Comroe, Jr.

ACKNOWLEDGMENT

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[††] Unfortunately, Neergaard worked with the equilibrium methods of Jäger (24) and Lenard (25) instead of methods that permitted changing the area of a surface film so as to alter the concentration of water-insoluble molecules such as lipids; Neergaard must have regarded the moist surface alveolar layer as water-soluble.

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events and product problems

Page ___ of ___

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FDA Use Only (Resp Care)

Triage unit
sequence #

A. Patient information

1 Patient identifier

In confidence

2 Age at time of event: ______
or
Date of birth:

3 Sex
☐ female
☐ male

4 Weight
____ lbs
or
____ kgs

B. Adverse event or product problem

1 ☐ Adverse event and/or ☐ Product problem (e.g., defects/malfunctions)

2 Outcomes attributed to adverse event (check all that apply)

☐ death ______ (mo/day/yr)
☐ life-threatening
☐ hospitalization – initial or prolonged
☐ disability
☐ congenital anomaly
☐ required intervention to prevent permanent impairment/damage
☐ other ______

3 Date of event (mo/day/yr)

4 Date of this report (mo/day/yr)

5 Describe event or problem

6 Relevant tests/laboratory data, including dates

7 Other relevant history, including preexisting medical conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1 Name (give labeled strength & mfr/labeler, if known)
#1
#2

2 Dose, frequency & route used
#1
#2

3 Therapy dates (if unknown, give duration) from/to (or best estimate)
#1
#2

4 Diagnosis for use (indication)
#1
#2

5 Event abated after use stopped or dose reduced
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

6 Lot # (if known)
#1
#2

7 Exp. date (if known)
#1
#2

8 Event reappeared after reintroduction
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

9 NDC # (for product problems only)
___ – ___ – ___

10 Concomitant medical products and therapy dates (exclude treatment of event)

D. Suspect medical device

1 Brand name

2 Type of device

3 Manufacturer name & address

4 Operator of device
☐ health professional
☐ lay user/patient
☐ other ______

5 Expiration date (mo/day/yr)

6
model # ______
catalog # ______
serial # ______
lot # ______
other #

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8 If explanted, give date (mo/day/yr)

9 Device available for evaluation? (Do not send to FDA)
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3 Occupation

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(Abstracts continued from Page 279)

associated with decreased exposure in the control groups. CONCLUSION: Such long-term maintenance of behavior change is highly unusual in the general behavioral science literature, let alone for addictive behaviors. We conclude that ETS exposure can be reduced and that a clinician-delivered treatment may provide substantial benefit.

Reducing Length of Stay for Patients Hospitalized with Exacerbation of COPD by Using a Practice Guideline—GK Kong, MJ Belman, S Weingarten. Chest 1997;111(1):89.

Clinical practice guidelines have been promoted as an effective way of reducing costs while maintaining quality care. OBJECTIVE: To study a practice guideline to shorten length of stay for patients hospitalized with exacerbation of COPD. METHODS: We retrospectively studied a practice guideline to identify patients who were at low risk of complications from their exacerbation of COPD and hence potentially suitable for early hospital discharge. We then prospectively studied the practice guideline using an alternate month intervention and control time series over a period of 12 months. RESULTS: The practice guideline was retrospectively studied in 250 consecutive patients hospitalized with exacerbation of COPD. Of the 250 patients, 237 patients (94.8%) were classified as low risk after 72 hours of hospitalization and were potentially suitable for discharge. In the prospective study, few patients (24 of 124 or 19%) were identified for implementation of the guideline. However, in those patients who were identified, length of stay was not statistically different. The data also showed that length of stay for both intervention and control groups had shortened over this time. CONCLUSION: Certain practice guidelines may appear efficacious in studies but may actually lack effectiveness when applied in clinical settings and may even increase costs. We demonstrated the importance of prospectively evaluating clinical practice guidelines before recommending them for widespread implementation.

Cytomegalovirus as a Primary Pulmonary Pathogen in AIDS—AB Waxman, SJ Goldie, H Brett-Smith, RA Matthay. Chest 1997;111(1):128.

In patients with AIDS, isolation of cytomegalovirus (CMV) from respiratory secretions is common. It is often found with other pathogens, which has led to debate regarding its role as a primary pulmonary pathogen. A retrospective investigation of patients with AIDS and CMV as a sole pulmonary isolate was performed in an attempt to describe their clinical presentation and course. All patients admitted to the hospital with pneumonia and with BAL or transbronchial biopsy (TBB) specimen positive for CMV between 1991 and 1994 were identified through a review of inpatient records. Inclusion criteria included positive CMV cultures from BAL, cytomegalic inclusion bodies from BAL or TBB, and thorough documentation of the absence of other pulmonary pathogens. Nine patients met the inclusion criteria for CMV pneumonitis. Seven were male and 2 were female, ages 26 to 44 years, and all had a history of opportunistic infections. Typical clinical presentation was characterized by increased respiratory rate, hypoxemia, and diffuse interstitial infiltrates. The mean CD4 count was 29.6 (± 22) cells/cubic millimeter, mean lactate dehydrogenase level was 414 (± 301) IU/L, and in 7 patients in whom CMV antigen was measured it was greater than 50 positive cells per 200,000 WBCs. Three untreated patients died of respiratory failure and 3 had autopsy confirmation of CMV pneumonia. Five patients were treated with anti-CMV therapy for at least 2 weeks, and all demonstrated improvement in symptoms, oxygen saturation, and chest radiograph. At 3 months follow-up, all 5 patients were asymptomatic with no pulmonary symptoms. At 6 months follow-up, 3 of the 5 patients remained asymptomatic; the other 2 died of other opportunistic infections. In at least these 9 patients, CMV represented a primary pulmonary pathogen. Patients who were treated responded quickly and were able to be discharged home from the hospital with marked improvement in their symptoms. We recommend that clinicians consider this diagnosis in the proper setting and consider treatment with anti-CMV therapy.

Applied PEEP during Pressure Support Reduces the Inspiratory Threshold Load of Intrinsic PEEP—NR MacIntyre, K-CG Cheng, R McConnell. Chest 1997;111(1):188.

OBJECTIVES: Mechanical ventilation in patients with obstructive airway disease (OAD) is associated with the development of dynamic hyperinflation and intrinsic positive end-expiratory pressure (PEEPi). One of the effects of this form of PEEPi is to act as an inspiratory threshold load that can produce ineffective breath triggering, dyspnea, and muscle fatigue. Recently it has been shown that applying PEEP in the ventilator circuit can reduce this imposed triggering load. We wished to investigate this further by studying patients with OAD being weaned with pressure support (PS) ventilation. Our first objective was to determine the prevalence and magnitude of this form of PEEPi in OAD patients who were clinically judged to be capable of triggering mechanical ventilatory breaths. Our second objective was to attempt to reduce the triggering load by applying circuit PEEP and then observe the response of patient-ventilator interactions during the patient-triggered, pressure-limited PS breath. DESIGN: Thirteen random patients with OAD who were receiving PS ventilation were studied by measuring airway pressures, airway gas flow, baseline esophageal pressure, esophageal pressure time products (PTP), and esophageal pressure changes before ventilator gas delivery began (ΔPes taken to represent PEEPi). Measurements were made at baseline and after stepwise increases in circuit PEEP up to the PEEPi. RESULTS: We found measurable PEEPi in all patients (average ± SD of 9.54 ± 4.3 cm H_2O) and it was > 10 cm H_2O in 7 patients. As would be predicted, we observed progressive reductions in PEEPi as applied PEEP was given. We also observed that the component of patient effort (PTP) related to overcoming PEEPi also decreased, but the PTP related to tidal volume (V_T) did not. The V_T associated with the set PS thus did not change with application of PEEP, nor did the breathing frequency. CONCLUSION: PEEPi is common in OAD patients receiving mechanical ventilatory support. The imposed triggering load from PEEPi can be offset to large extent by circuit PEEP approaching the baseline PEEPi. Although total patient effort substantially falls with applied PEEP, the patient effort that combine with PS to effect V_T does not.

The Effects of Motion on the Performance of Pulse Oximeters in Volunteers (Revised publication)—SJ Barker, NK Shah. Anesthesiology 1997;86(1)101.

BACKGROUND: Pulse oximetry is considered a standard of care in both the operating room and the postanesthetic care unit, and it is widely used in all critical care settings. Pulse oximeters may fail to provide valid S_{pO_2} data in various situations that produce low signal-to-noise ratio. Motion artifact is a common cause of oximeter failure and loss of accuracy. This study compares the accuracy and data dropout rates of 3 current pulse oximeters during standardized motion in healthy volunteers. METHODS: Ten healthy volunteers were monitored by 3 different pulse oximeters: Nellcor N-200, Nellcor N-3000, and Masimo SET (prototype). Sensors were placed on digits 2, 3, and 4 of the test hand, which was strapped to a mechanical motion table. The opposite hand was used as a stationary control and was monitored with the same pulse oximeters and an arterial cannula. Arterial oxygen saturation was varied from 100% to 75% by changing the inspired oxygen concentration. While S_{pO_2} was both constant and changing, the oximeter sensors were connected before and during motion. Oximeter errors and dropout rates were digitally recorded continuously during each experiment. RESULTS: If the oximeter was functioning before motion began, the following are the percentages of time when the instrument displayed an S_{pO_2} value within 7% of control: N-200 = 76%, N-3000 = 87%, and Masimo = 99%. When the oximeter sensor was connected after the beginning of motion, the values were N-200 = 68%, N-3000 = 47%, and Masimo = 97%. If the alarm threshold was chosen S_{pO_2} less than 90%, then the positive predictive values (true alarms/total alarms) are N-200 = 73%, N-3000

= 81%, and Masimo = 100%. In general, N-200 had the greatest S_{pO_2} errors and N-3000 had the highest dropout rates. CONCLUSIONS: The mechanical motions used in this study significantly affected oximeter function, particularly when the sensors were connected during motion, which requires signal acquisition during motion. The error and dropout rate performance of the Masimo was superior to that of the other 2 instruments during all test conditions. Masimo uses a new paradigm for oximeter signal processing, which appears to represent a significant advance in low signal-to-noise performance.

Are Chest Radiographs and Electrocardiograms Still Valuable in Evaluating New Pediatric Patients with Heart Murmurs or Chest Pain?—JM Swenson, DR Fischer, SA Miller, GJ Boyle, JA Ettedgui, LB Beerman. Pediatrics 1997; 99(1):1.

OBJECTIVES: To determine the usefulness of electrocardiography (ECG) and chest radiography (CXR) in evaluation of patients referred to the pediatric cardiologist for the evaluation of heart murmur or chest pain. DESIGN: In this prospective study, 106 consecutive outpatients were categorized with no heart disease, possible heart disease, or definite heart disease based on history and physical examination; they then underwent ECG and CXR. Studies were reviewed and the examining cardiologist could change the diagnosis and order an echocardiogram. SETTING: Academic pediatric cardiology practice. RESULTS: In patients thought to have no heart disease, the diagnosis was changed to definite heart disease in 4 solely on the basis of abnormal CXR or ECG. In 25 patients thought to have possible heart disease, the diagnosis was changed to no heart disease (7) or definite heart disease (5) after review of the CXR and ECG. All 25 patients diagnosed with definite heart disease had this confirmed by abnormal CXR (2), ECG (3), both abnormal CXR and ECG, or echocardiogram (18). CONCLUSIONS: ECG and CXR helped diagnose heart disease in 4 patients thought to have no heart disease, helped to rule out lesions in 7 patients with possible heart disease, helped diagnose heart disease in 5 patients thought to have possible heart disease, and helped confirm heart disease in 9 patients. In these days of cost containment, routine ECG and CXR continue to be valuable tools for the pediatric cardiologist in evaluation of patients with heart murmurs or chest pain.

Effects of Proportional Assist Ventilation on Inspiratory Muscle Effort in Patients with Chronic Obstructive Pulmonary Disease and Acute Respiratory Failure—VM Ranieri, S Grasso, L Mascia, S Martino, T Fiore, A Brienza, R Giuliani. Anesthesiology 1997;86(1)79.

BACKGROUND: Acute respiratory failure may develop in patients with chronic obstructive pulmonary disease because of intrinsic positive end-expiratory pressure (PEEPi) and increased resistive and elastic loads. Proportional assist ventilation is an experimental mode of partial ventilatory support in which the ventilator generates flow to unload the resistive burden (flow assistance: FA) and volume to unload the elastic burden (volume assistance: VA) proportionally to inspiratory muscle effort, and PEEPi can be counterbalanced by application of external PEEP. The authors assessed effects of propordonal assist ventilation and optimal ventilatory settings in patients with chronic obstructive pulmonary disease and acute respiratory failure. METHODS: Inspiratory muscles and diaphragmatic efforts were evaluated by measurements of esophageal, gastric, and transdiaphragmatic pressures. Minute ventilation and breathing patterns were evaluated by measuring airway pressure and flow. Measurements were performed during spontaneous breathing, continuous positive airway pressure, FA, FA + PEEP, VA, VA + PEEP, FA + VA, and FA + VA + PEEP. RESULTS: FA + PEEP provided the greatest improvement in minute ventilation (89 ± 3%) and dyspnea (62 ± 2%). The largest reduction in pressure time product per breath of the respiratory muscles and diaphragm (44 ± 3% and 33 ± 2%, respectively) also was observed during FA + PEEP condition. When VA was added to this setting, a reduction in respiratory rate (50 ± 3%), an increase in inspiratory time (102 ± 6%), and a further reduction in pressure time product per minute (65 ± 2% and 64% for the respiratory muscles and diaphragm, respectively) was observed. However, values of pressure time product per liter

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RESPIRATORY CARE September 1997
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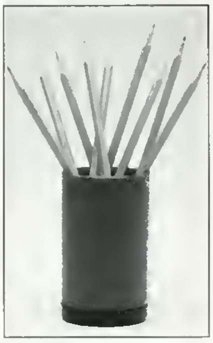

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of minute ventilation during FA + VA + PEEP did not differ with those observed during FA + PEEP condition. Worsening of patient-ventilator interaction and breathing asynchrony occurred when VA was implemented. CONCLUSIONS: Application of PEEP to counterbalance PEEPi and FA to unload the resistive burden provided the optimal conditions in such patients. Ventilator over-assistance and patient-ventilator asynchrony was observed when VA was added to this setting. The clinical use of proportional assist ventilation should be based on continuous measurements of respiratory mechanics.

Pulmonary Effect of Inhaled Furosemide in Ventilated Infants with Severe Bronchopulmonary Dysplasia—A Kugelman, M Durand, M Garg. Pediatrics 1997;99(1):71.

BACKGROUND: When administered parenterally, furosemide, a loop diuretic, results in improved lung compliance and decreased airway resistance in infants with bronchopulmonary dysplasia (BPD). However, furosemide-induced diuresis results in hypokalemia, chloride deficiency, hypercalciuria, nephrocalcinosis, and rickets. In patients with asthma, inhaled furosemide has recently been demonstrated to inhibit the bronchoconstrictive effects of exercise, cold air hyperventilation, and antigen challenge. We hypothesized that inhaled furosemide will result in improved pulmonary mechanics in ventilated infants with BPD and will prevent the systemic complications of parenteral furosemide. OBJECTIVE: To determine the efficacy and safety of a single dose of inhaled furosemide on pulmonary mechanics in infants with severe BPD who are ventilator dependent at 21 days of age. DESIGN & METHODS: A randomized, double-blind, crossover study was performed on 9 infants with BPD, each serving as his own control. Each patient was randomized to receive an aerosol dose of furosemide (1 mg/kg in 2 mL of saline) or placebo (2 mL of saline) on the first day of the study and the other agent the following day of the study. Pulmonary mechanics were measured before and 1 and 2 hours after the inhalation using the Pulmonary Evaluation and Diagnostics System. RESULTS: Gestational age (mean ± SEM) was 29 ± 1 weeks; birthweight was 1.1 ± 0.1 kg, age at study was 47 ± 6 days; and weight at study was 1.8 ± 0.2 kg. There was no significant change in the pulmonary function measurements before treatment and 1 or 2 hours after treatment with either placebo or furosemide. Baseline and 2-hour values were: dynamic compliance (mL/cm H_2O/kg): 0.46 ± 0.03 to 0.50 ± 0.03 (placebo) and 0.50 ± 0.02 to 0.51 ± 0.02 (furosemide); dynamic resistance (cm H_2O/L/s): 118 ± 9 to 106 ± 7 (placebo) and 111 ± 8 to 105 ± 7 (furosemide); and tidal volume (mL/kg): 8.6 ± 0.5 to 8.9 ± 0.5 (placebo) and 8.9 ± 0.2 to 9.4 ± 0.3 (furosemide). CONCLUSION: We conclude that, under the conditions of our study, a single dose of 1 mg/kg inhaled furosemide does not improve the pulmonary mechanics in ventilator-dependent infants with severe BPD.

Clinical Practice Guidelines in Pediatric and Newborn Medicine: Implications for Their Use in Practice—TA Merritt, D Palmer, DA Bergman, PH Shiono. Pediatrics 1997;99(1):100.

Clinical practice guidelines are becoming pervasive in pediatrics and newborn medicine. They have spanned a wide range of primary care practice parameters from treating otitis media with effusion, to performing complex surgery for congenital heart disease, and management of respiratory distress syndrome and coordinating discharge from the neonatal intensive care unit. Administrators believe that using clinical practice parameters reduces health care costs, improves quality of care, and limits malpractice liability. Practice parameters and guidelines have grown in use because powerful interests—third-party payers, insurers, and health maintenance organizations, as well as hospital administrators bent on reducing variable costs of care and contracting for capitated care—champion their development, implementation, and monitoring. Economic credentialing of physicians with excessive variances without risk-adjusting for other than average patients is problematic and remains unchecked partly because of the fundamental characteristics of the evolving health care industry in which costs are more easily measured than quality. For highly autonomous physicians this standardization of medical decision making may represent a difficult transition into corporate practice by realigning traditional values of the doctor-patient relationship. However, because guidelines are almost certainly here to stay, pediatricians and neonatologists need to think critically about how their content and method of implementation, monitoring, and modification may influence medical teaching and decision making in the future. If guidelines are introduced primarily as a cost-savings or containment tool that ignores the impact on the quality of care and restricts necessary care for infants and children, especially those with chronic illness or who are developmentally at risk, then neonatologists and pediatricians must be quick and determined to challenge the potentially damaging use of practice parameters or guidelines. Furthermore, there are many medicolegal implications of guideline implementation that may not

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favor physicians and leave to hospitals, insurers, and ultimately the courts decisions regarding evidence-based practice. In this review article, we pay special attention to the guidelines developed in newborn medicine. We discuss why and how guidelines are developed and critically evaluate the available evidence describing potential benefits and drawbacks of guidelines in general. There are legal implications to the implementation of guidelines, and guidelines may increase provider susceptibility to malpractice allegations. Neonatologists and pediatricians should critically analyze the following questions when guidelines are being developed: Are clinical practice parameters the most effective means to reduce the costs of health care, or improve the quality of health care services while reducing the need for and protecting physicians from malpractice suits? Or do clinical practice guidelines more closely resemble an audit system developed by health care organizations, insurers, and others including government-sponsored health care to appease powerful interests-with limited evidence for promise and perhaps potential negative cost, quality, and malpractice liability implications? In pediatric and newborn medicine there is limited evidence that guidelines have achieved the desired goals and further analysis of their process of care and the costs of implementation is warranted.

Death in the Intensive Care Nursery: Physician Practice of Withdrawing and Withholding Life Support—SN Wall, JC Partridge. Pediatrics 1997;99(1):64.

OBJECTIVE: To determine the frequency of selective nontreatment of extremely premature, critically ill, or malformed infants among all infant deaths in a level III intensive care nursery (ICN) and to determine the reasons documented by neonatologists for their decisions to withdraw or withhold life support. METHODS: This was a descriptive study based on review of the medical records of all 165 infants who died at a university-based level III ICN during 3 years. We determined whether each death had occurred despite the use of all available technologies to keep the infant alive or whether these were withheld or withdrawn, thereby leading to the infant's death. We also determined whether neonatologists documented either "futility" or "quality of life" as a reason to limit medical interventions. RESULTS: One hundred sixty-five infants died among the 1,609 infants admitted during the study period. One hundred eight infant deaths followed the withdrawal of life support, 13 deaths followed the withholding of treatment, and 44 deaths occurred while infants continued to receive maximal life-sustaining treatment. For 90 (74%) of the 121 deaths attributable to withholding of withdrawal of treatment, physicians cited that death was imminent and treatment was futile. Quality-of-life concerns were cited by the neonatologists as reasons to limit treatment in 62 (51%). Quality of life was the only reason cited for limiting treatment for 28 (23%) of the 121 deaths attributable to withholding or withdrawal of treatment. CONCLUSIONS: The majority of deaths in the ICN occurred as a result of selective nontreatment by neonatologists, with few infants receiving maximal support until the actual time of death. Neonatologists often documented that quality-of-life concerns were considered in decisions to limit treatment; however, the majority of these decisions were based on their belief that treatment was futile. Prospective studies are needed to elucidate the determinants of neonatologists' practice decisions of selective nontreatment for marginally viable or damaged infants.

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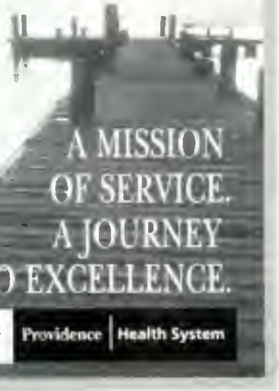

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Calendar of Events

AARC & AFFILIATES

April 1–4—Indiana, Kentucky, and Ohio Societies

24th Annual Region II for Respiratory Care Meeting, "Winning the Game of Respiratory Care," at the Hyatt Regency Hotel and the Albert Sabin Convention Center, Cincinnati, Ohio.

Contact: 1-800-691-3046, mailbox #1, or http://www.bright.net/~dsibb/Reg2rc.htm on the Internet.

April 2–4—Tennessee Society

Annual convention and exhibition, "Wilderness Zone: Transitions for Tomorrow," at the Park Vista Hotel overlooking the Smokey Mountains, in Gatlinburg, Tennessee.

Contact: Colleen Schabacker at (615) 384-1731.

April 15–16—Pennsylvania Society

32nd Annual Southeast District Seminar, "Happy 50th Respiratory Care," at the Airport Ramada, Essington, Pennsylvania.

Contact: Angie Herstine at (609) 784-0340 or Ann Cusano at (215) 646-7300 ext 428.

April 25—Texas Society's Alamo District, the University of Texas Health Science Center at San Antonio (UTHSCSA), and Wilford Hall Medical Center

Seminar at the San Antonio Municipal Auditorium and Holiday Inn Riverwalk North, San Antonio, Texas.

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Contact: Respiratory Care, UTHSCSA, (210) 567-3706.

May 20–21—Connecticut Society

Super Symposium, "Respiratory Care: The Magic Is in Your Hands," at the Four Points Sheraton, Waterbury, Connecticut.

Contact: Nancy Apruzzese, Director, Respiratory Care Services, (860) 224-5264.

June 5–6—Maine Society

Maine Event Conference at the Marriot in South Portland, Maine.

CRCE: 5/day, all Category 1.

Contact: Bobbie Crockett, (207) 262-1632.

June 11–13—Texas Society

Annual convention, "The Star of Texas," at the Marriott Riverwalk Hotel, San Antonio, Texas. Management Training Institute seminar is scheduled.

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Contact: TSRC at (972) 680-2454.

OTHER MEETINGS

March 26–27—Johns Hopkins Hospital Department of Respiratory Care

Two-day seminar, "Lectures in Adult and Pediatric Respiratory Care," Baltimore, Maryland. Lunch is provided both days.

Contact: Jennifer Lee at (410) 955-9277.

April 17–18—California Society for Pulmonary Rehabilitation

Annual conference, "Bridging the Waters—A Continuum of Care," at the Capitol Plaza Holiday Inn, Sacramento, California.

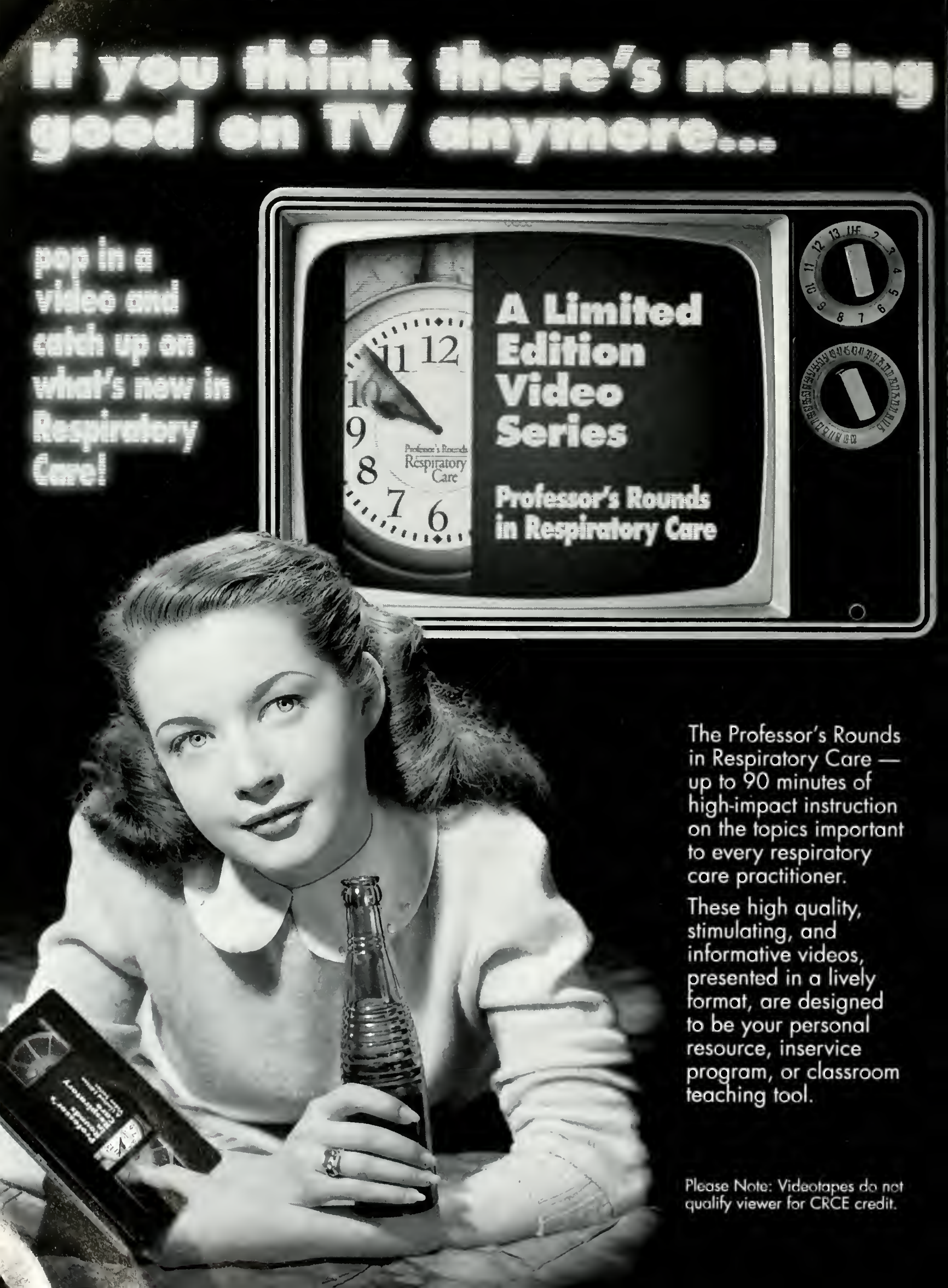
Conference registration: $185. Preconference session, "Components of a Successful Pulmonary Rehab Program," is April 16; registration: $75.

Contact: Barbara Rife, CSPR Conference Co-Chair, Mercy San Juan Hospital, Pulmonary Rehabilitation, 6501 Coyle Ave, Carmichael CA 95608, (916) 537-5299.

May 16–21—American Lung Association/American Thoracic Society (ALA/ATS)

Annual International Conference in San Francisco, California.

Contact: 1997 International Conference, ALA/ATS, 1740 Broadway, New York NY 10019-4374.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Identifies the different forms of invasive and noninvasive hemodynamic monitoring, their main complications, and appropriate levels. Discusses how positive end-expiratory pressure and other therapies affect hemodynamic parameters.

Weaning from Mechanical Ventilation — Item VC23
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Pulmonary Rehabilitation — Item VC24
Learn the components of a comprehensive pulmonary rehabilitation program, selecting appropriate candidates, identifying and setting both short- and long-term patient goals. Also covers assessment of a patient's progress and follow-up.

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Presents the basic principles of aerosol administration and the efficacy and use of metered-dose inhalers versus nebulizers. Also instructs in the administration and assessment of the effects of bronchodilators during mechanical ventilation and the techniques for the correct patient use of aerosol devices.

Prevention of Postoperative Atelectasis and Pneumonia — Item VC26
Identifies the patients at increased risk of postoperative atelectasis and pneumonia and how to decrease the risk of nosocomial pneumonia. Also covers its pathogenesis and the roles of chest physiotherapy, bronchoscopy, and other therapies in managing acute lobar atelectasis.

Patient-Focused Care — Item VC31
Learn about the assumptions at work when employing the model, its ramifications, the changing roles for employees in ancillary service departments, and the multifaceted nature of the model.

Application of Positive Airway Pressure Without Intubation — Item VC32
Covers short-term application in the inpatient setting in the treatment of acute, life-threatening conditions and elective, long-term application in home care. Includes a discussion of bi-level positive airway pressure via the BiPAP® device.

Therapist-Driven Protocols in Respiratory Care — Item VC33
Learn how therapist-driven protocols can assist practitioners in providing better patient care and containing costs. Provides an overview to the challenges to implementing protocols and how to gain the support of key players in the health care team.

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Reviews the determinants of tissue oxygenation in the critically ill patient and the techniques available for assessment. Also discusses "supply dependency" of tissue oxygen utilization in ARDS and how it affects management.

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Unconventional Methods for Adult Oxygenation and Ventilation Support — Item VC36
Provides an overview of new and experimental techniques for adult oxygenation and ventilation support. Discusses the techniques' theoretical rationales, methods of application, and experimental evidence of effectiveness.

Tuberculosis: Implications for Patients and Practitioners — Item VC41
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Discusses therapist-driven protocol (TDP) implementation strategies, research, staff selection, training, obstacles, attitudes of other staff, and the results of TDP implementation.

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Blood Gas and Related Measurements: Laboratory versus Bedside Devices — Item VC44
Presents the various means of obtaining blood gas information, invasive and noninvasive devices, the need for such devices, and when they should be used.

Hospital Operational Restructuring and Respiratory Care — Item VC45
Reviews restructuring products, their implementation results, their relationship to respiratory care, and ways practitioners and managers can influence them.

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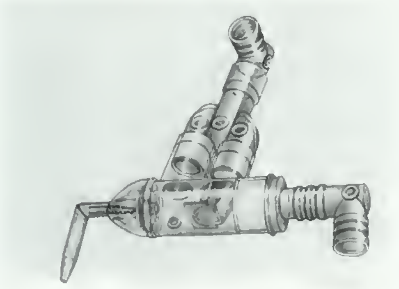
A VHA Satellite Network Production from the American Association for Respiratory Care

New Products & Services

News releases about new products and services will be considered for publication in this section. There is no charge for these listings. Send descriptive release and glossy black and white photographs to RESPIRATORY CARE Journal, New Products and Services Dept, 11030 Ables Lane, Dallas TX 75229-4593.

INFECTION CONTROL PRODUCTS. Afassco Inc announces a line of infection control products that protects health care workers from blood borne pathogens. According to the company, the products—Stat-5, a hard-surface disinfectant; SteriDerm, an antimicrobial hand spray; SteriWash, an antimicrobial hand soap; and Medi-Wipes, single-use antimicrobial towelettes—protect against hepatitis, human immunodeficiency virus, and tuberculosis. The products can be used in first aid, blood borne pathogen, and biohazard kits. For details about each product, circle Reader Service Number 160.



AEROSOL SYSTEM. AMICI adds the Swirler™ Aerosol System to its line of products. The Swirler is designed for use with radioaerosols. The company claims that the plastic device delivers aerosols of efficient particle size to the airways and alveoli for use in radioaerosol studies and diagnostic procedures. The Swirler also allows medications to bead up for gravity rainout and readministers the aerosol to the lung. The device is designed for single-patient use and has a variety of accessories to accommodate different applications. Circle Reader Service Number 161 for more details.

CYLINDER & LIQUID PORTABLES CART. A lightweight cart, designed to carry both liquid oxygen portables and E oxygen gas cylinders, debuts with Andonian Cryogenics Inc. The company says the cart provides space for both E cylinders and liquid portables and is useful for home health care personnel. The cart has interlocking nylon straps for cylinder use and a 5 1/2 in. × 8 1/4 in. basket for portables use. Information is available; circle Reader Service Number 162.

POLYSOMNOGRAPHY SYSTEM. Bio-logic Systems Corporation introduces the Sleepscan Traveler® monitoring system. The system—designed for in-home monitoring—uses a battery and weighs < 2 pounds. The company says that the monitor is programmable, available in 8-, 19-, or 27-channel platforms, and features a built-in pulse oximeter and calibration and impedance checks. Data can be viewed simultaneously on 1 screen. Other features include the data analysis software and Smart-Pack™ data compression. For details or to learn more about the ambulatory electroencephalogram testing system, circle Reader Service Number 163.

ELECTRODES. Burdick includes Heart-Line DuraFoam and Comfort Tape wet-gel electrodes in its product line. The electrodes are designed for short-term monitoring and use in cardiac stress testing, Holter monitoring, and SAECG applications, Burdick claims. The electrodes feature soft, pliable materials that mold to body contours, low-chloride gel and gentle adhesives for decreased skin irritation, and a stabilization ring that anchors the electrode to the body for stable ECG baselines and reduced motion artifact. Circle Reader Service Number 164 for more information.

PEDIATRIC PEAK FLOW METER & TRAINER. To help young children produce consistent peak flow scores, Clement Clarke offers the miniaturized Aerodynamic Flow System (AFS) low-range peak flow meter and Windmill Trainer. The peak flow meter is designed to capture the lower peak flows of young patients. When used with the Windmill

Trainer, training children to use the peak flow meter is easier, the company says. The trainer clips to the Mini AFS meter and can be positioned close to the mouthpiece, where a small blow turns the sails; as the child progresses the Windmill may be moved further away to build technique. For details from the company, circle Reader Service Number 165.

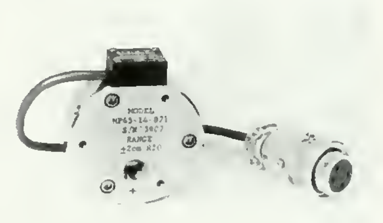
ENDOTRACHEAL TUBE/BITE BLOCK. The Thumb, developed by the Thumbs Up Group, is an endotracheal tube (ETT)/bite block device designed to stabilize ETTs and provide comfort to mechanically ventilated patients. According to the manufacturer, The Thumb is designed for better oral hygiene in intubated patients, eliminates sticky tape, and helps prevent external oral-dermal breakdown. The system consists of a combination bite block-ETT holder in bright orange, 36 in. of umbilical tape, and 2 half-moon-shaped, nonporous sponges. The device can be used in pediatric patients who are older than 8 years and in adults. To learn more about the system, circle Reader Service Number 166.

POCKET POLYGRAPH. The NightOwl™ Pocket Polygraph is now included in Respironics Inc's line of products. The NightOwl offers diagnostic sleep recording in a compact package, allows up to 15 channels for recording patient data, and uses low power from a rechargeable NiCad battery for 10 hours of use. The polygraph's software allows data to be viewed in real time and correlates data for patient reports via IBM-type computers. The polygraph is made of solid-state components, has a built-in impedance meter, and is capable of oximetry. Circle Reader Service Number 167 for details.

ASTHMA MEDICATION. Zeneca Pharmaceuticals releases a new asthma medication. According to Zeneca, the U.S. Food & Drug Administration (FDA) has cleared Accolate® (zafirlukast) tablets for the preventive and chronic treatment of asthma in adults and children 12 years of age and older. The oral medication is the first leukotriene receptor antagonist to receive FDA clearance; and the drug works by blocking the effects of leukotrienes, natural substances in the body that may contribute to asthma symptoms. To receive information about indications, contraindications, dosing requirements, and a complete fact sheet, circle Reader Service Number 168.

BEDSIDE TEST. A rapid bedside test for cardiac troponin I—CARDIAC STATUS™—is announced by Spectral Diagnostics Inc. The company says that CARDIAC STATUS is approved by the U.S. Food & Drug Administration for point-of-care testing for troponin I, a marker for myocardial cell injury. The test can be used in emergency departments, critical care units, and other hospital settings. The test uses whole blood, serum, or plasma. Positive results may appear within minutes, depending on the level of troponin I in the blood. Negative results take 15 minutes to confirm. The test is read by a simple line scheme in the test window. For indications, contraindications, and a more detailed product guide, circle Reader Service Number 169.

PRODUCT BROCHURE. Polyfoam Packers Corporation offers a new product brochure for mail-order and home care health services. According to the company, the brochure includes information about Thermosafe® insulated storage chests, field carriers, specimen transporters, therapeutic cold packs, and more. To receive a free brochure, circle Reader Service Number 170.



DIFFERENTIAL PRESSURE TRANSDUCER. A low-range variable reluctance differential pressure transducer—the MP 45-871—is released by Validyne Engineering. The transducer is used for pulmonary measurements in conjunction with the Validyne carrier demodulators. According to the company, the transducer ranges ±2 to ±880 cm H_2O, offers a pressure cavity volume of 0.16 cc, and uses a volumetric displacement of 0.016 cc. The device is made of stainless steel, monitors pulmonary flow, volume, and pressure, and supports electronics for interface with chart recorders or computers. Information about Carrier demodulators and the MP 45-871 is available from the manufacturer. Circle Reader Service Number 171.

Manuscript Preparation Guide

General Information

RESPIRATORY CARE welcomes original manuscripts related to respiratory care and prepared according to these Instructions. Manuscripts are blinded and reviewed by professionals who are experts in their fields. Authors are responsible for all aspects of the manuscript and receive galleys to proofread before publication. Each accepted manuscript is copyedited so that its message is clear and it conforms to the Journal's style. Published papers are copyrighted by Daedalus Inc and may not be published elsewhere without permission.

Editorial consultation is available at any stage of planning or writing. On request, specific guidance is provided for all publication categories. These Instructions and related materials are available. Write to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593, call (972) 243-2272, or fax (972) 484-6010.

Publication Categories & Structure

Research Article: A report of an original investigation (a study). It includes a Title Page, Abstract, Introduction, Methods, Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figures, and Figure Captions.

Evaluation of Device/Method/Technique: A description and evaluation of an old or new device, method, technique, or modification. It has a Title Page, Abstract, Introduction, Description of Device/Method/Technique, Evaluation Methods, Evaluation Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figures, and Figure Captions.

Case Report: A report of a clinical case that is uncommon, or was managed in a new way, or is exceptionally instructive. All authors must be associated with the case. A case-managing physician must either be an author or furnish a letter approving the manuscript. Its components are Title Page, Abstract, Introduction, Case Summary, Discussion, References, Tables, Figures, and Figure Captions.

Review Article: A comprehensive, critical review of the literature and state-of-the-art summary of a pertinent topic that has been the subject of at least 40 published research articles. Title Page, Outline, Introduction, Review of the Literature, Summary, Acknowledgments, References, Tables, Appendices, and Figures and Captions may be included.

Overview: A critical review of a pertinent topic that has fewer than 40 published research articles.

Update: A report of subsequent developments in a topic that has been critically reviewed in this Journal or elsewhere.

Point-of-View Paper: A paper expressing personal but substantiated opinions on a pertinent topic. Title Page, Text, References, Tables, and Illustrations may be included.

Special Article: A pertinent paper not fitting one of the foregoing categories may be acceptable as a Special Article. Consult with the Editor before writing or submitting such a paper.

Editorial: A paper drawing attention to a pertinent concern; it may present an opposing opinion, clarify a position, or bring a problem into focus.

Letter: A signed communication about prior publications in this Journal or about other pertinent topics. Tables and illustrations may be included. Mark "For publication."

Blood Gas Corner: A brief, instructive case report involving blood-gas values—with Questions, Answers, and Discussion.

Drug Capsule: A mini-review paper about a drug or class of drugs that includes discussions of pharmacology, pharmacokinetics, and pharmacotherapy.

Graphics Corner: A brief case report incorporating waveforms for monitoring or diagnosis—with Questions, Answers, and Discussion.

Kittredge's Corner: A brief description of the operation of respiratory care equipment—with information from manufacturers and editorial comments and suggestions.

PFT Corner: Like Blood Gas Corner, but involving pulmonary function tests.

Cardiorespiratory Interactions. A case report demonstrating the interaction between the cardiovascular and respiratory systems. It should be a patient-care scenario; however, the case—the central theme—is the systems interaction. CRI is characterized by figures, equations, and a glossary. See the March 1996 issue of RESPIRATORY CARE for more detail.

Test Your Radiologic Skill: Like Blood Gas Corner, but involving pulmonary medicine radiography and including one or more radiographs, may involve imaging techniques other than conventional chest radiography.

Review of Book, Film, Tape, or Software: A balanced, critical review of a recent release.

Preparing the Manuscript

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Center main section headings on the page and type them in capital and small letters (eg, Introduction, Methods, Results, Discussion). Begin subheadings at the left margin and type them in capital and small letters (eg, Patients, Equipment, Statistical Analysis).

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Article in a journal carrying pagination throughout volume:

1. Rau JL, Harwood RJ. Comparison of nebulizer delivery methods through a neonatal endotracheal tube: a bench study. Respir Care 1992;37(11):1233-1240.

Article in a publication that numbers each issue beginning with Page 1:

2. Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

3. American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to promote retrieval.)

4. Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986;89(3, Suppl):139S-143S.

Abstract in journal: (Abstracts citations are to be avoided. Those more than 3 years old should not be cited.)

5. Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990:35(11): 1087-1088.

Editorial in journal:

6. Enright P. Can we relax during spirometry? (editorial). Am Rev Respir Dis 1993;148(2):274.

Editorial with no author given:

7. Negative-pressure ventilation for chronic obstructive pulmonary disease (editorial). Lancet 1992;340(8833):1440-1441.

Letter in journal:

8. Aelony Y. Ethnic norms for pulmonary function tests (letter). Chest 1991;99(4):1051.

Paper accepted but not yet published:

9. Hess D. New therapies for asthma. Respir Care (year, in press).

Personal author book: (For any book, specific pages should be cited whenever possible.)

10. DeRemee RA. Clinical profiles of diffuse interstitial pulmonary disease. New York: Futura, 1990:76-85.

Corporate author book:

11. American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group, 1977.

Chapter in book with editor(s):

12. Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott, 1977.

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Notices

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RESPIRATORY CARE Special Issues

The April 1997 issue of the Journal features papers and discussions from the Consensus Conference: Noninvasive Positive Pressure Ventilation held in Vail, Colorado, October 4-6, 1996. The conference was chaired by Neil R MacIntyre MD and Dean R Hess PhD RRT. Authors include David J Pierson MD, Robert M Kacmarek PhD RRT, John R Bach MD, W Gerald Teague MD, Nichols R Hill MD, Gordon D Rubenfeld MD, Richard G Wunderink MD, Dean R Hess PhD RRT, and Robert E Turner RRT.

* * *

September 1997 issue of the Journal will be devoted to education—respiratory care formal programs and curriculum, continuing and in-service education, and patient education. Original studies, state-of-the-art reviews, teaching features, reviews of instructional materials, and other areas of education are encouraged. Send your unpublished manuscripts for peer review. For more details, call Kris Williams, Assistant Editor, (972) 406-4665.

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Helpful Web Sites

American Association for Respiratory Care
http://www.aarc.org

National Board for Respiratory Care
http://www.nbrc.org

Applied Measurement Professionals Inc
http://www.applmeapro.com

Food and Drug Administration
http://www.fda.gov

Center for Devices and Radiological Health
http://www.fda.gov/cdrh/index.html

Tuberculosis Information
http://www.umdnj.edu/ntbc

The Lambda Beta Society
The National Honor Society for the Profession of Respiratory Care

For information about the Lambda Beta Society—
the National Honor Society for the
Profession of Respiratory Care,
contact the Society Office at
1701 W Euless Blvd, Suite 200, Euless TX 76040
(817)283-4269.

John H "Jack" Emerson, AARC Honorary Member Dies

John H "Jack" Emerson, founder and president of J H Emerson Co in Cambridge, Massachusetts, and an American Association for Respiratory Care Honorary Member since 1982, died Tuesday, February 4, 1997. He was 90. Emerson improved the design for the 'iron lung' and was recognized as a leader in the development of medical devices, receiving 35 patents during his lifetime. He is survived by his wife, Madeleine, and two sons, William and George.

FDA Offers Regional Workshops

To help implement the *Medical Devices: Current Good Manufacturing Practice Final Rule/Quality System Regulation*, the Food and Drug Administration (FDA) is offering regional workshops. For information about a workshop near you, contact the representative in your area.

Northeast Region—George R Walden, 850 Third Ave, Brooklyn NY 11232, (718) 965-5300 ext 5528.

Mid-Atlantic Region—Joseph X Phillips, 900 U.S. Customhouse, 2nd & Chestnut St, Philadelphia PA 19106, (215) 597-0537.

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The National Board for Respiratory Care—1997 Examination Dates and Fees

Examination	Examination Date	Examination Fee	
CRTT Examination	March 8, 1997	$ 100	(new applicant)
	Application Deadline: January 1, 1997	60	(reapplicant)
	July 12, 1997	100	(new applicant)
	Application Deadline: May 1, 1997	60	(reapplicant)
	November 8, 1997	100	(new applicant)
	Application Deadline: September 1, 1997	60	(reapplicant)
RRT Examination	June 7, 1997	100	Written only (new applicant)
	Application Deadline: February 1, 1997	60	Written only (reapplicant)
		110	CSE only (all applicants)
	December 6, 1997	210	Both (new applicant)
	Application Deadline: August 1, 1997	170	Both (reapplicant)
CPFT Examination	June 7, 1997	110	(new applicant)
	Application Deadline: April 1, 1997	80	(reapplicant)
RPFT Examination	December 6, 1997	160	(new applicant)
	Application Deadline: September 1, 1997	130	(reapplicant)
Perinatal/Pediatric Respiratory Care Specialty Examination	March 8, 1997	160	(new applicant)
	Application Deadline: November 1, 1996	130	(reapplicant)

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